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THE ROENTGEN DIAGNOSIS OF INTRATHORACIC NEOPLASMS[1]

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WHILE there is undoubtedly an actual increase in the incidence of intrathoracic newgrowths, as attested by the study of postmortem statistics (Staehelin, 1, Barron, 2, Berblinger, 3), the increase is in some measure relative and due to improved methods of diagnosis, notably the X-ray and bronchoscope. Improved surgical procedures and roentgen therapy in malignant disease of the lungs are factors which have increased the general interest.

From the point of view of roentgen diagnosis pulmonary neoplasms fall into two

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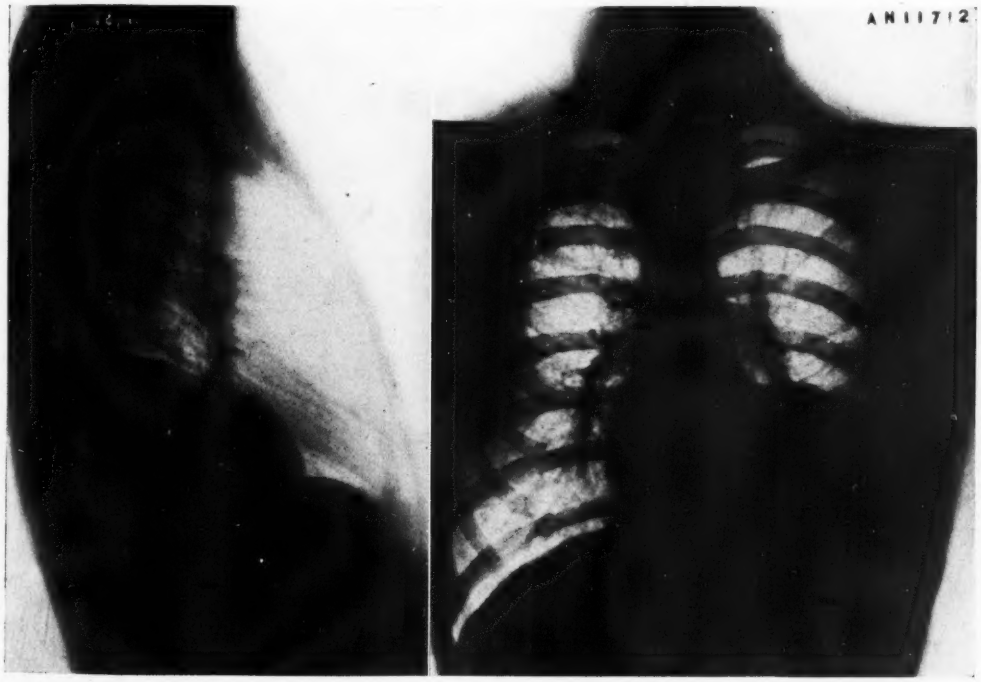

Fig. 1-*A* Cyst in the lateral posterior portion of the left lower lobe. Male, 22 years of age, patient of Dr. Chevalier Jackson. Cough of 6 months' duration; hemoptysis 3 months prior to admission.

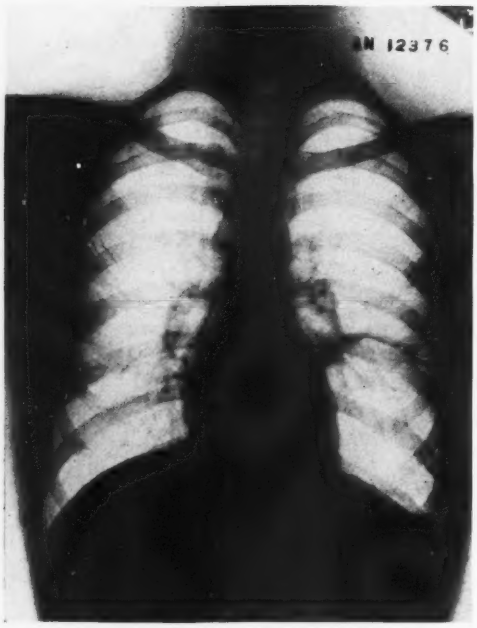

Fig. 1-*B*. Same case as shown in Figure 1-*A*. Fluid level in the cyst after bronchoscopy by Dr. Gabriel Tucker, guided fluoroscopically by Dr. Willis F. Manges. Pathologic report of contents by Dr. C. J. Bucher: dark reddish fluid, many micro-organisms, no echinococcous hooklets, flukes, or ova.

groups: first, those which are recognized readily and permit of accurate diagnosis; second, those in which the diagnosis is made with difficulty and then only by a consideration of all data. There is no sharp line of distinction between the two groups, though, in general, benign and metastatic lesions and those arising from the mediastinum are diagnosed more directly than are primary malignant changes.

In the first group, presenting definitive characteristics, are cysts, teratomata, many tumors of lymphatic origin, and certain types of metastases.

Cysts may be cryptogenic in origin or due to infestation: in the former instance they are usually single; in the latter they may be single or multiple, depending upon the type of parasite or the severity of the invasion. The cyst of congenital origin (Figs. 1 and 2) is often an accidental finding, though it may be associated with hemoptysis, the expectoration of pus, or pain in the chest. It may occupy any portion of the lung and varies greatly in size; its wall delimits it sharply from the rest of the lung and in both the antero-posterior and lateral projections it presents a round homogeneous density. Degenerative changes in the cyst wall may progress even to calcification (Fig. 3).

Cysts due to infestation are sometimes discovered accidentally, but very often attention is called to their presence by pain in the chest, a hemorrhage, or the expectoration of their contents after the rupture of the cyst wall and expulsion into a bronchus. Heuser (4) says that hydatid cysts may occur in any portion of the lung, they may be single or multiple, and vary greatly in size. Rupture into the pleural cavity may obscure the underlying hydatid disease by the development of an empyema. These cysts are round, with an homogeneous content, and present no evidence of fluid level before rupture and drainage through a bronchus.

Dermoids and teratomata are cyst-like in character but are differentiated from the simple cyst by the presence of calcified material within them; on the films the even density of the cyst has a denser body within it (Fig. 4). Pathologically, dermoids and teratomata differ from each other only in the complexity of their structure, and roentgenographically they cannot be distinguished. The possibility of their undergoing malignant change makes their recognition of some importance.

In the tumors of lymphatic origin, lymphomata, thymomata, lymphosarcomata, lymphadenomata, and those of the Hodgkin's group are found. In many instances, in spite of the fact that they evidence a great variety of forms, they are characteristic; at least, it is possible to say that there

Fig. 2. Cyst near the root-area in the anterior and middle portion of the right lung. Male, 54 years of age, patient of Dr. Chevalier Jackson. Cough for many years.

Fig. 3. Same case as shown in Figure 2. Oblique view, showing calcification of the cyst wall.

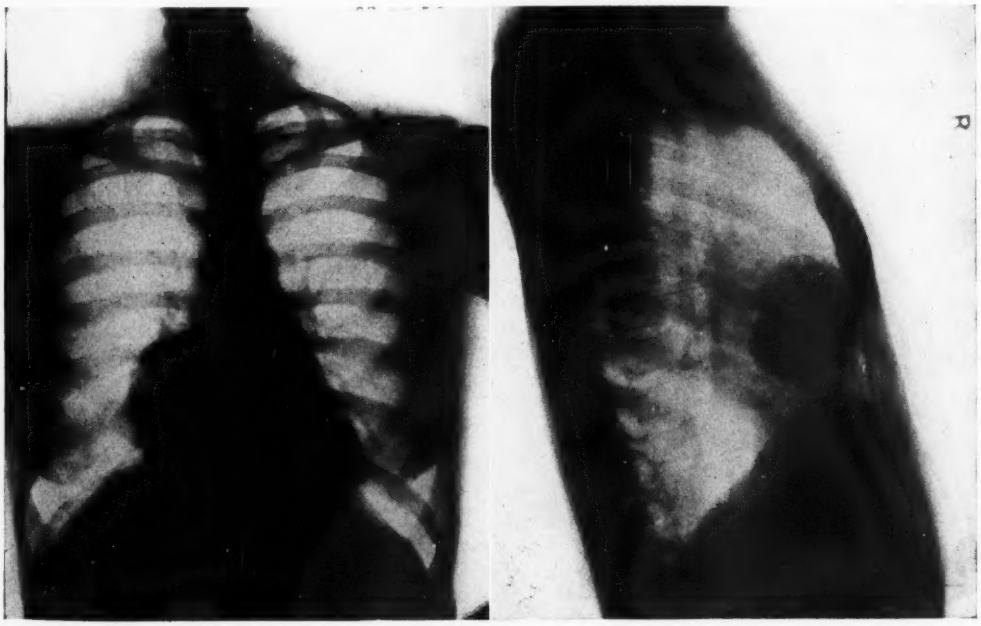

Fig. 4. Dermoid cyst or teratoma in the lower medial anterior portion of the right lung. A calcified deposit may be seen within the cyst. Female, 34 years of age, patient of Dr. Elmer H. Funk. Tumor accidentally discovered during a general examination, asymptomatic. No change over an interval of two years.

Fig. 5. Hodgkin's disease. Male, 31 years of age, patient of Dr. Hiram R. Loux. Cough, of increasing severity, of 6 months' duration, accompanied by progressive dyspnea, night sweats, loss of weight and strength.

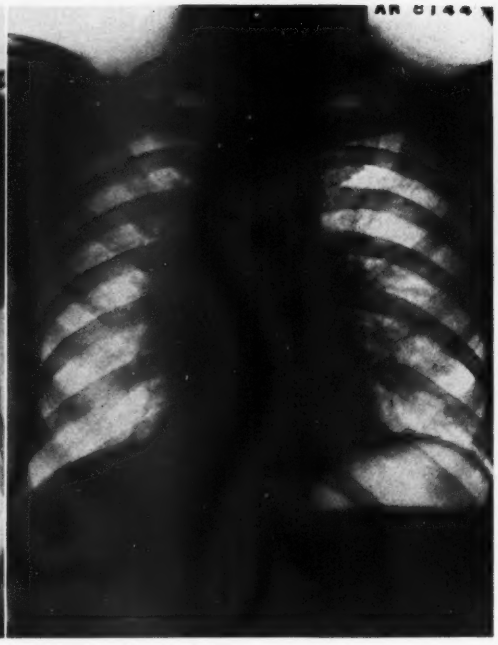

Fig. 6. Same case as shown in Figure 5 after a course of X-ray therapy during which symptoms disappeared and the patient regained his strength and weight. Later, recurrence terminated fatally.

is a newgrowth present. Arising at the root-area or along the mediastinal aspect of the lung, they vary from the size of a walnut to great amorphous masses completely filling one side (and in some instances almost all of both sides) of the thorax. Presenting an even density, they may be round or oval in outline and their connection with the mediastinal structures is readily recognized. These lymph tumors are almost as sharply defined as cysts, but usually they are somewhat fuzzier. Occasionally, because of pressure, they produce collapse of a portion of a lobe and then the density of the tumor blends with the atelectatic lung in such a way that its own sharp outline is lost. In his celebrated monograph Adler (5) says that lymphosarcomata never undergo cavity formation. Roentgen therapy in most instances causes these tumors to lessen in size and sometimes to disappear, though their disappearance is usually but temporary and eventually they end fatally. Figures 5 and 6 present such a case. This rapid response to the roentgen ray serves to distinguish them from other tumors, notably the extrabronchial carcinomata which they sometimes resemble in structure, and from substernal enlargements of the thyroid and aortic aneurysm, with which, in rare instances, they may be confused.

Metastasis from other organs may take place in the tracheobronchial lymph nodes at the root-area and upper mediastinum (Fig. 10), or directly in the parenchyma (Figs. 7, 8, and 9). Metastases to the nodes appear as masses at the hilum. They cannot be distinguished roentgenologically from some primary lymphatic tumors, but they usually do not disappear so promptly when exposed to the action of the roentgen ray.

Fig. 7. Nodular metastasis. Male, 52 years of age, patient of Dr. Charles F. Nassau. Kidney removed 18 months earlier because of tumor. Histologic diagnosis by Dr. B. L. Crawford: adenocarcinoma of the kidney.

Fig 8. Nodular metastasis. Male, 21 years of age, patient from the service of Dr. John H. Gibbon. Mass in the axilla; pain in the leg.

Parenchymal pulmonary metastases are of four types, the nodular, miliary, infiltrative, and massive.

In the nodular, one finds sharply circumscribed nodules of varying size scattered throughout the pulmonary fields. Occasionally there is but a single nodule; more often they are multiple. They are of an even density, and, while they have the sharpness of cysts, they are of a lesser density. Multiple shadows of this type are pathognomonic of metastasis (Figs. 7 and 8).

The miliary type presents many of the characteristics of the nodular (Fig. 9) except that the metastatic masses are smaller and evenly distributed throughout the lungs.

In the infiltrative and massive types the findings are not so certainly indicative of metastatic malignancy. In the former there is a radial increase in the markings arising at a root-area (Fig. 11): the shadows are usually sharper than in inflammatory proc-

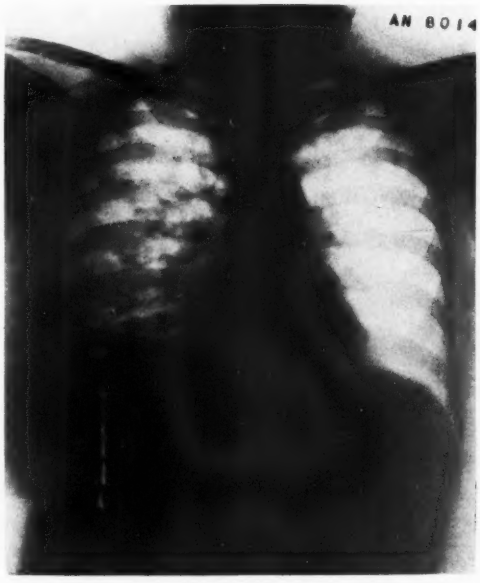

Fig. 9. Miliary metastasis. Female, 33 years of age, patient of Dr. S. Jaffe. Both lungs involved from carcinoma of the left breast.

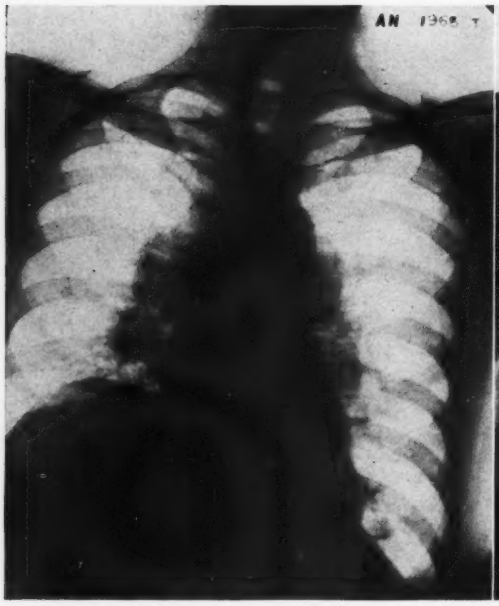

Fig. 10. Multiple metastases to lymph nodes at the root-areas; marked elevation of the diaphragm on the right. Male, 63 years of age, patient of Dr. Willis F. Manges. Metastasis to cervical nodes also, and to the mandible.

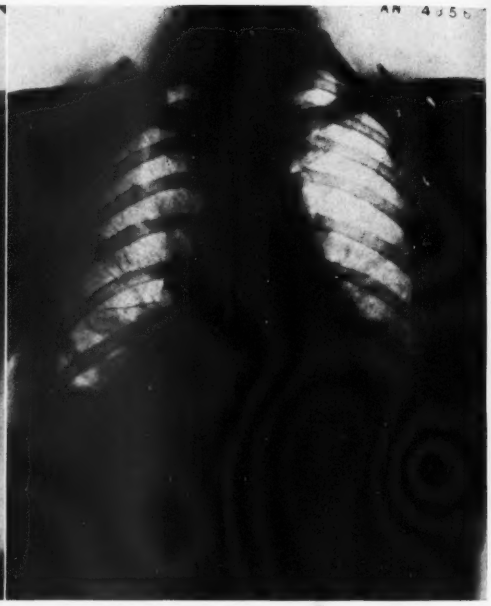

Fig. 11. Metastatic infiltration radiating from the right hilum into the lower half of the right lung; diaphragm elevated. Female, 55 years of age, patient of Dr. Edward J. Klopp. Carcinoma of the right breast.

esses. When this type of increase is associated with a primary newgrowth it should be looked upon as metastatic. Massive metastatic changes are due to consolidation of a lobe or part of a lobe (Fig. 12), and may be associated with effusion into the pleural cavity.

Infiltrative and massive metastatic changes occupy a position comparable to the finding of blood-tinged fluid in the pleural cavity. While the bloody aspirate alone is strong presumptive evidence of metastasis, it becomes practically absolute when removed from an individual suffering from malignant disease of some other organ. Alone, infiltrative and massive changes may only suggest metastasis, but when they are associated with a primary carcinoma in some other organ they should be considered and treated as metastatic until proven otherwise.

Primary pulmonary malignancy is diagnosed less readily than are the benign and metastatic tumors. The roentgen appearance is determined by many factors, one of which is the point of origin—whether it is in the bronchi or in the interstitial tissues. McCrae, Funk, and Jackson (6) quote Passler (7) as saying that 87 per cent of all lung carcinomata are of bronchial origin, and they advocate applying this term, "bronchial carcinomata," to these, and reserving the term "pulmonary carcinoma" for instances in which the possibility of bronchial origin of the newgrowth has been excluded by the most careful bronchoscopic study.

Extrabronchial growths are not readily differentiated from lymphatic mediastinal tumors: they are, however, usually more dense and have somewhat sharper borders. They are less apt to be of a regular round or oval pattern and may be wavy and irregular in outline. In some instances they are ill-defined and present, as in Figure 13, an

Fig. 12. Massive metastases, with infiltration in lower portion of the left lung. Female, 54 years of age, patient of Dr. Frederick J. Kalteyer. Inoperable carcinoma of the left breast, tumor of 10 years' duration: retraction of the soft tissues of the left axilla may be distinguished.

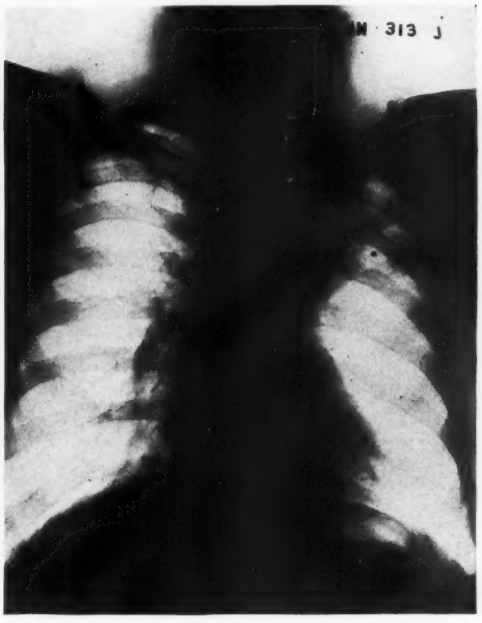

Fig. 13. Extrabronchial neoplasm in the upper portion of the left chest at the mediastinal border, difficult to differentiate from tumor of lymphatic origin. Male, 35 years of age, patient of Dr. Chevalier Jackson. Paroxysmal cough of 11 months' duration; expectoration of pus; loss of weight; dizziness. At bronchoscopy. Dr. Jackson could find no endobronchial growth.

irregular margin. Carcinomata of this type do not respond so readily to roentgen therapy as do the lymphatic tumors, and in this failure we have a therapeutic test to differentiate the two types. It is sometimes difficult to distinguish these extrabronchial tumors from substernal prolongation of the thyroid, or, when they are in close relation to the large vessels, from aneurysm; in the latter instance the absence of expansile pulsation and the negative Wassermann may be the deciding factors.

Occasionally, as in Figure 14, these extrabronchial tumors resemble those arising from the bronchi.

Endobronchial tumors are divided by Ewing (8) into three groups according to their origin: first, those arising from the bronchial epithelium; second, those from the mucous glands; third, those originating in the alveoli. Ewing says that even at postmortem it is possible to classify only the early and less malignant cases; the extensive and rapidly growing so merge that their origin cannot be determined.

Symptomatically there is little that is outstanding in these patients. Cough is the most frequently occurring single symptom —McCrae, Funk, and Jackson found it in all fourteen of the cases they reported. Expectoration was a symptom in twelve, pain in eleven, and hemorrhage and dyspnea occurred in eight.

The roentgen picture of primary bronchial malignancy is determined by the stage at which the tumor is found and its point of origin. The signs are indirect, due to obstructive phenomena, and direct, due to the shadow of the tumor. That the diagnosis is not always easy is emphasized by Golden (9), Kornblum (10), Hyde and Holmes (11), Hirsch (12), and Kirklin and Pater-

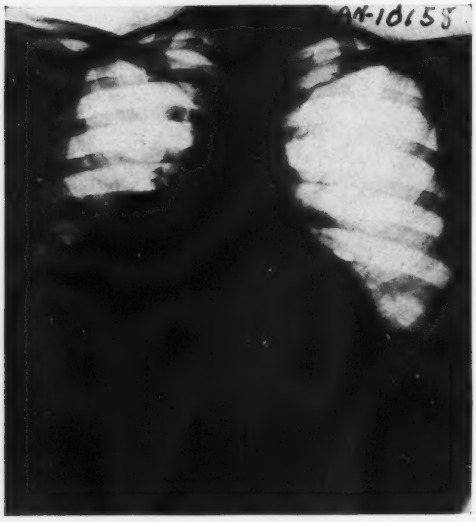

Fig. 14. Extrabronchial neoplasm in the lower portion of the right lung, seeming to radiate from the hilum; atelectasis, with elevation of the diaphragm. Female, 31 years of age, patient of Dr. Chevalier Jackson. Dyspnea of increasing severity of 3 months' duration, with pain in the back and neck; vertigo; nausea, and loss of weight. Dr. Jackson reported from bronchoscopy a broadened carina, "infiltrative lesion, probably malignant, involving and closing the right main bronchus."

Fig. 15. Extrabronchial neoplasm in the right upper lobe. Female, 70 years of age, patient of Dr. Chevalier Jackson. Tumor is undoubtedly of long standing because of the marked retraction of the right chest. The trachea is narrowed, its rings calcified.

son (13). Kirklin and Paterson (13) say: "The major roentgenologic characteristics of bronchial tumors are twofold: density in the hilum, and atelectatic or bronchiectatic appearance." Hirsch (12) in his study found that, "the roentgen appearance is due to the following changes: (1) tumor; (2) stenosis-atelectasis; (3) inflammation; (4) necrosis; (5) cirrhosis; (6) pleuritis—fibrinous and exudative; (7) adenopathy." From these statements it will be readily seen that the factors influencing the appearance of primary carcinoma of the bronchus are complex.

At its onset an epithelioma of the bronchus should act as a foreign body and produce signs pointed out by Manges (14) and by Iglauer (15). The degree of these functional obstructive phenomena is determined by the site of origin: the nearer the bifurcation the tumor arises the more pronounced these disturbances. It has never been my good fortune to study a bronchial carcinoma in the very earliest stages, that is, before it cast a direct shadow and was producing only obstructive signs, but I have seen a patient with bronchial papillomata and it is possible to draw an analogy.

The patient, a woman of 39 years, was referred to Dr. Jackson's Bronchoscopic Clinic complaining of a progressively increasing wheezing dyspnea from which she had suffered for nine years. The difficulty in breathing was particularly marked when lying down. A diagnosis of asthma had been made repeatedly. In the first roentgenograms there was a slight increase in the pulmonary markings in both lower lobes to which no clinical significance was attached. The patient was not examined fluoroscopically, and even had she been

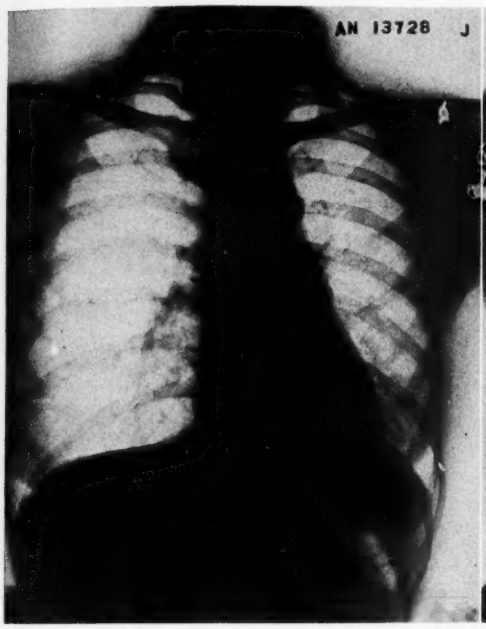

Fig. 16-*A*. Film made at inspiration. Endobronchial neoplasm: papillomata and granulomata involving the left bronchus. Increase in the pulmonary markings in the left lower lobe; displacement of the heart to this side and elevation of the diaphragm. At bronchoscopy, previous to this exposure, Dr. Aucoin had removed papillomatous masses and granulation tissue from the right bronchus. Female, 39 years of age, patient of Dr. Chevalier Jackson. Paroxysmal cough, dyspnea, and occasional expectoration of blood for 4 weeks.

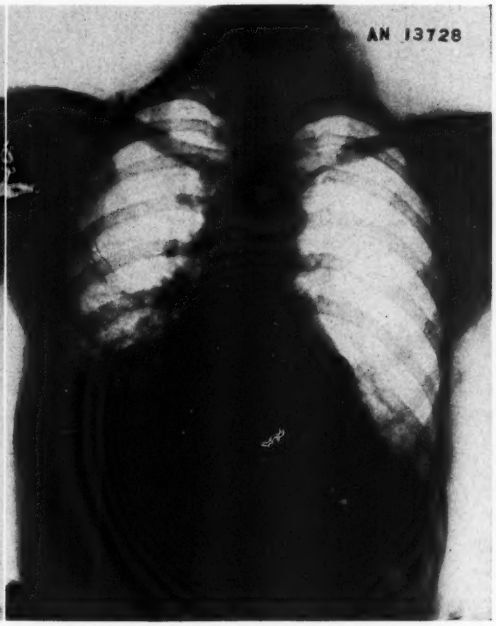

Fig. 16-*B*. Same case as shown in Figure 16-*A*. Film made at expiration. Endobronchial neoplasm: papillomata and granulomata involving the left bronchus. Obstructive emphysema of the left lung; displacement of the heart to the right; depression of the diaphragm on the left; left side of the chest distended. This film was made before Dr. Aucoin had removed the obstructing masses from the left bronchus.

probably nothing distinctive would have been observed. At the first bronchoscopy Dr. Aucoin found both main bronchi filled with papillomatous growths and granulomata which, because of the attendant bleeding, could be removed only from one side. After operation the patient was again radiographed and a decided difference was found in the two lungs. On the side from which the growths had been removed there was a free egress of air, the diaphragm rose, the chest contracted, and the lung emptied. On the opposite side there was an obstructive emphysema, the diaphragm remained depressed with expiration, the heart was displaced to the sound side because of the unequal intrapulmonary pressures on the two sides, and air could not leave the lung (Figs. 16-*A* and 16-*B*). Subsequently, the growths were removed from the second bronchus and on re-examination it was found that both lungs emptied equally well and that there were no obstructive phenomena.

In an early case of epithelioma of the bronchus one may expect these same signs, and the nearer the origin of the growth to the bifurcation the more marked will be the findings. With continued growth of the tumor the lumen of the bronchus will be gradually narrowed and eventually occluded. With complete occlusion there follows absorption of the air distal to the growth, with collapse of this portion of the lung accompanied by findings similar to those described

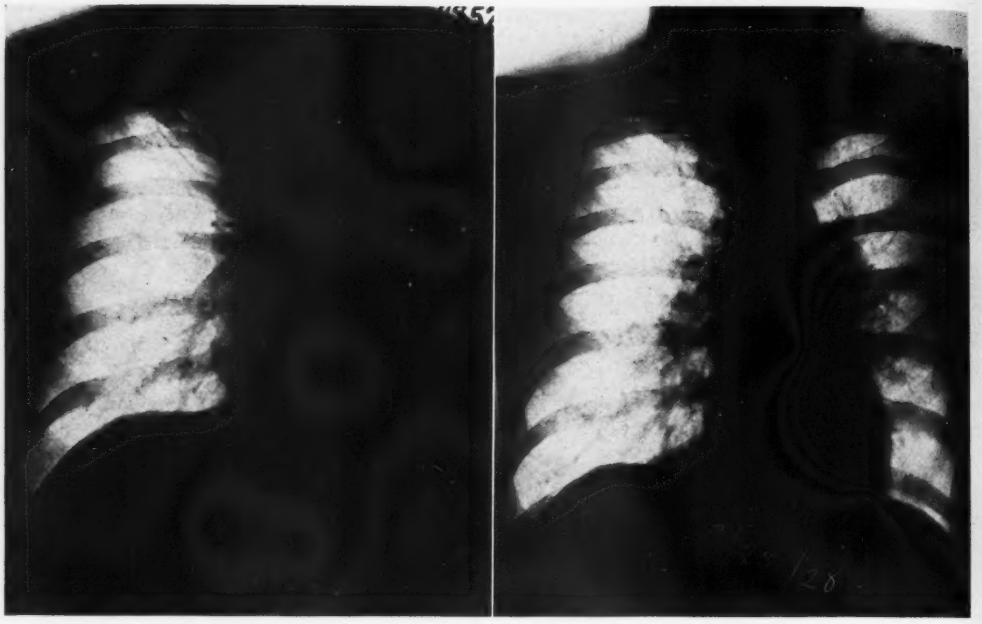

Fig. 17-*A*. Endobronchial neoplasm: carcinoma of the left bronchus. Film made Feb. 25, 1928. Complete atelectasis of the left lung, with displacement of the heart and mediastinal structures to the left. Male, 40 years of age, patient of Dr. Chevalier Jackson. Recurring attacks of massive collapse of the left lung accompanied my hemoptysis, pain, dyspnea, and cough. Patient had coughed up polyp-like mass, histologically diagnosed as carcinoma, before admission, with relief of collapse. At bronchoscopy, Dr. Louis H. Clerf removed tissue reported, after histologic study by Dr. B. L. Crawford, to be carcinoma of the bronchus.

Fig. 17-*B*. Same case as shown in Fig. 17-*A*. Endobronchial neoplasm: carcinoma of the left bronchus. This film was made March 24, 1928—compare it with Figure 17-*A*, made earlier. Atelectasis of the left lung has disappeared after successive bronchoscopies by Dr. Louis H. Clerf.

by Manges (16) in the case of completely occluding bronchial foreign bodies. These signs are diminished density, progressing to complete opacity on the occluded side, displacement of the heart and other mediastinal structures to this side, accompanied by elevation of the diaphragm. Figures 17-*A* and 17-*B* show an instance in which a recurring carcinoma of the bronchus produced an intermittent atelectasis, relieved either by the patient coughing up some of the tumor tissue or by its bronchoscopic removal. In Figure 23 is shown a permanent atelectasis of the left lung due to a long-standing tumor of the bronchus.

In addition to the foregoing indirect signs of newgrowth of the bronchus we have the direct signs due to the mass and changes secondary to its presence—obstructive and pressure phenomena, inflammatory changes, and metastasis.

In bronchial carcinoma the mass usually appears as a nodule at the hilum (Fig. 17-*B*). It may attain great size (Fig. 18) or remain small and localized. The density of these masses varies: often they are faint, sometimes they approach in density the consolidation of lobar pneumonia. In outline they are practically always more irregular than are the benign tumors or the tumors of lymphatic origin, due to the fact that the tumor frequently grows irregularly and its shadow outlines the projections. The appearance of the mass is in some instances influenced by the effect its pressure has on

Fig. 18. Endobronchial neoplasm: carcinoma of the right bronchus. Nodular mass at the right root-area; atelectasis of the lower lobe. Male, 36 years of age, patient of Dr. Chevalier Jackson. Cough; expectoration of blood-stained mucus; pain in the right chest of 5 months' duration. At bronchoscopy, Dr. Louis H. Clerf removed a piece of tissue diagnosed histologically by Dr. B. L. Crawford as carcinoma of the bronchus.

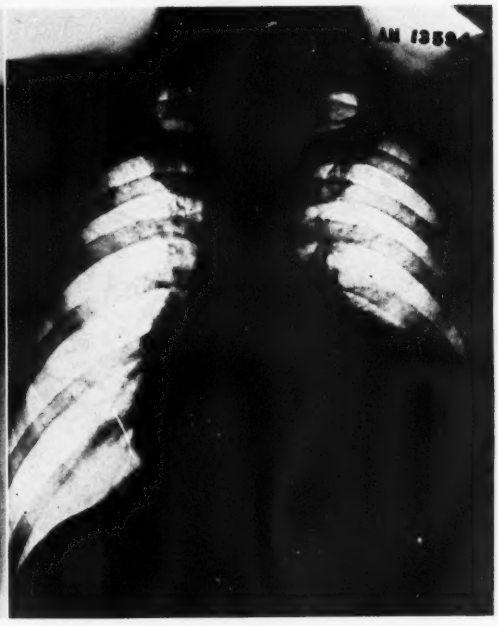

Fig. 19. Endobronchial neoplasm: carcinoma. Mass in the lower portion of the left half of the chest; diaphragm elevated on the left but heart displaced to the right. Male, 43 years of age, patient of Dr. Chevalier Jackson. Symptoms of one year's duration—cough, expectoration of blood-stained mucus, loss of 50 pounds in weight. Tissue removed at bronchoscopy reported by Dr. B. L. Crawford to be carcinoma.

neighboring tissue, as in Figure 18, in which there is probably an area of atelectasis in addition to the tumor. The hilum is not the only site of these tumors: they are sometimes a little distance from it and fairly well surrounded by sound lung. Occasionally the lesion is infiltrative in type and difficult to distinguish from inflammatory or metastatic infiltration (Fig. 19). Occlusion of the bronchus, whether due to the endobronchial mass or to pressure on neighboring bronchi, interferes partially or wholly with drainage and leads to localized areas of drowned lung. Secondary to this poor drainage there follows infection of the area and the development of suppurative foci in these tissues of locally lessened resistance. The result may take on the form of Figure 21, in which the foci are of a bronchiectatic type, or it may progress to extensive cavity formation, as in Figure 22, in which the condition simulates one of multiple abscesses.

Metastasis to other portions of the lungs occurs occasionally with tumors of this type and leads to their diagnosis. This condition obtains in Figure 21, in which there is a mass in the upper portion of the left lung and suppurative changes in the lower medial portion. Spread to the pleura may result in the development of fluid in the pleural cavity and bring about the signs of effusion.

CONCLUSIONS

1. Pulmonary cysts of congenital origin, teratomata, dermoids, and unruptured

Fig. 20. Endobronchial neoplasm: carcinoma. Area of increased density in the lower portion of the left lung, inflammatory in type. Male, 42 years of age, patient of Dr. Edward J. Klopp. Cough, expectoration of mucus and blood, pain in the left chest, loss of weight. At necropsy, tumor of the left lung was found, with central necrosis, reported by Dr. B. L. Crawford to be "carcinoma of the left lung, probably primary in the bronchus, with metastasis to the lymph nodes."

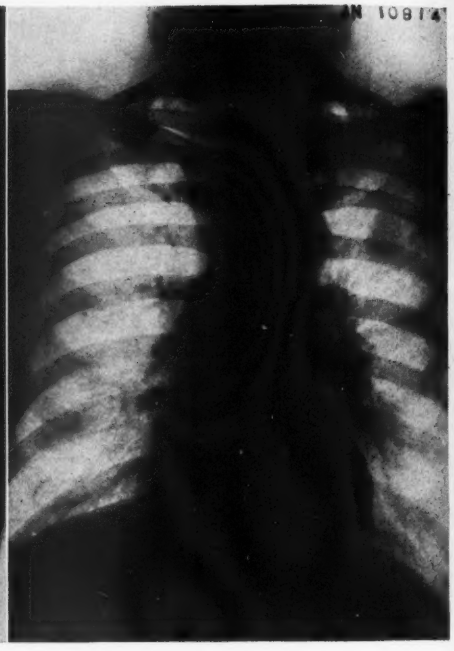

Fig. 21. Endobronchial neoplasm: carcinoma. Mass at the left root-area, increased markings in left lower lobe, with metastatic nodule in upper portion of the lung. Male, 53 years of age, patient from the service of Dr. Hobart A. Hare. Cough, expectoration, pain in the left chest and night sweats of 3 months' duration, and loss of weight. At autopsy, Dr. F. W. Konzelmann found a carcinoma of the left bronchus, with metastasis to the mediastinal nodes.

hydatid cysts present quite characteristic roentgen appearances.

2. Mediastinal tumors of lymphatic origin are also fairly characteristic; they respond well to roentgen therapy, and an exposure to a therapeutic dose of X-ray may make possible a differential diagnosis between these tumors, substernal thyroid, and extrabronchial carcinoma.

3. Metastases of some varieties, notably the nodular and the miliary, are readily diagnosed.

4. Infiltrative and massive changes in the lungs, while not always definitive of themselves, are pathognomonic when associated with a known primary malignant tumor of some other organ.

5. Extrabronchial (pulmonary) carcinomata, while they usually present characteristics which establish them as new-growths, may be confused with mediastinal lymphatic tumors.

6. Endobronchial (bronchial) carcinomata present the greatest problem in the diagnosis of intrathoracic tumors. The signs which they present depend upon their age and the point of origin.

7. The direct sign of endobronchial carcinoma is a mass due to the presence of a tumor, to which collapse, infection, or metastasis may be traced as secondary phenomena. Indirect signs are obstructive phenomena similar to those of bronchial foreign body.

Fig. 22. Endobronchial neoplasm: carcinoma, with abscess formation. Large fluid-containing cavity in the middle portion of the right lung. Male, 51 years of age, patient of Dr. Chevalier Jackson. Cough, slight expectoration of mucus, dyspnea, loss of 40 pounds in weight. At bronchoscopy, Dr. Louis H. Clerf removed tissue reported carcinoma by Dr. B. L. Crawford.

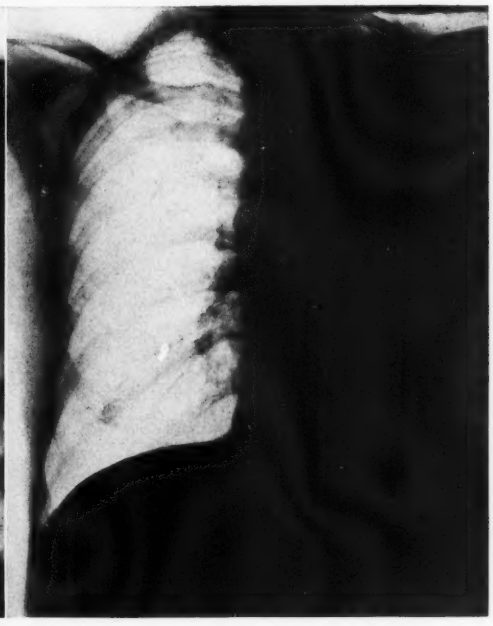

Fig. 23. Endobronchial neoplasm: carcinoma of the bronchus. Complete atelectasis of the left lung, with displacement of the heart and mediastinal structures to this side. Male, 60 years of age, patient of Dr. Chevalier Jackson. Dyspnea and hoarseness of 5 months' duration; loss of weight. Tissue removed at bronchoscopy by Dr. Louis H. Clerf was diagnosed as carcinoma of the bronchus by Dr. C. J. Bucher.

8. A more widespread recognition of the increasing incidence of bronchial carcinoma and a knowledge of the resulting changes should lead to earlier diagnosis and better response to roentgen therapy.

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DISCUSSION

Dr. E. L. Jenkinson (Chicago): Referring to Dr. Farrell's paper, I might mention findings in lung tumors which are not pathognomonic but are often an aid in making a diagnosis. It is not uncommon in bronchogenic carcinoma to find an area of atelectasis beyond the tumor, due to pressure causing the lung beyond to become atelectatic. The smaller bronchi are closed, allowing no air to enter. The air remaining is absorbed through the circulation. Following the atelectasis, an abscess may form. I saw two definite abscesses with definite fluid levels not long ago in a bronchogenic carcinoma.

I think it might be apropos to speak of bone metastases following carcinoma of the lung: they occur a good deal oftener than most of us think. Not long ago, at one of the hospitals with which I am associated, the pathologist wrote an article quoting five cases of bone metastases following carcinoma of the lung. We had an opportunity to radiate four of these patients before they died, one of them not more than a week before death. This man had had bone changes for three years previous to his death. I think Dr. Thomas and Dr. Blaine and Dr. Hirsch reported this case at the American Medical Association meeting in Washington. There were bone changes in the ulna and radius of one forearm. Sections were made and Wassermann tests were made. At no time was a definite diagnosis determined upon. We could not find anything in the chest cavity a week before the patient died, but at autopsy a small tumor at the junction of the bronchi was found, and our pathologist thought that all those changes in the bones were secondary to the primary carcinoma of the lung. The primary idea of the paper mentioned was to prove or disprove the possibility of so-called endothelioma (Ewing's tumor). Our pathologist maintains that the bone changes are all secondary to carcinoma of the lungs.

Dr. C. A. Hedblom (Chicago): The evidence that malignant disease of the lung is on the increase seems incontrovertible. It is based on thousands of necropsy examinations extending over a period of several decades. There is no adequate explanation, to my knowledge. The most plausible explanation so far advanced for the apparent increase in the last few years was the hemolytic streptococcus epidemic of 1918-1920. The greatest weakness of this explanation is the fact that more than 75 per cent of the carcinomatous tumors occur in men, while there was no disproportion in the sex incidence of the epidemic. Regardless of etiology, it is of the greatest importance, since the condition is on the increase, that we be on the alert for it and that we make every possible effort to establish an early diagnosis, both with a view to excluding other conditions and because of the possibility of radical curative treatment. The chief service that the roentgenologic department can render in the diagnosis is in establishing the presence of pathology in the lungs, and that is a very great service. At the same time, we must recognize that the diagnosis of malignant disease is fraught with great difficulties. The roentgenologic findings are simulated by so many other conditions, such as encapsulated empyema, encapsulated serous effusion, dermoids, abscess of the lung, etc.

The diagnosis can be established with certainty only microscopically. The sputum should be examined with greatest care for fragmentary tissue. Occasionally malignant cells can be found in aspirated pleural effusion. Biopsy of fragments removed at bronchoscopy or of tissue removed at ex-

ploratory thoracotomy will establish the diagnosis. Finding of carcinoma in excised glands furnishes us the basis for a presumptive diagnosis of carcinoma of the lung if there is definite pathology there and if no evidence can be found of a primary malignancy elsewhere. Rarely benign tumors such as fibroma and chondroma simulate carcinoma, but resemble more sarcoma of the chest wall.

In case of early carcinoma in the periphery or middle portion of the lung a lobectomy is indicated and offers some chance of cure. A few such three- to five-year cures are on record.

I have done an exploratory thoracotomy on three patients during the last year, but found the growth inoperable in all cases because of the fact that the hilus was involved. I inserted radon needles, which seemed to check the progress of the growth. One of these patients remained remarkably comfortable, attending to her household duties up to a very short time before her death.

I should like to emphasize the importance of keeping the possibility of malignant disease, particularly carcinoma, in mind in differential diagnosis.

Dr. L. T. LeWald (New York): I am a member of the Registry for Lung Tumors of the American Society of Thoracic Surgery. Now I think that blank forms will be sent to each member of this Society so that he may register his lung tumors as bone tumors are registered. I hope that we will really accomplish something in the definite registration and diagnosis of lung tumors, as much as—or, I hope, more than—has been accomplished by the bone registry, for example.

Dr. Farrell (closing): The important thing clinically in the diagnosis of intrathoracic neoplasms is undoubtedly the recognition that a malignant tumor exists as an entity. If the physician will remember this, and if the roentgenologist will recognize a change, and, unable to make a positive diagnosis, refer the patient to some one for bronchoscopic study, the early recognition of these tumors will undoubtedly increase. In early diagnosis lies the hope of treatment. I have seen in the last five years, five cancers of the lung which have been markedly improved by treatment. One of these patients was a girl, a photograph of whom Dr. Jackson gave me just before I left, with instructions that, if I could, I should exhibit her as illustrating the low grade malignancy of many of these tumors, and the way in which many of them respond to roentgen therapy. This girl had suffered for a year or a year and a half with thoracic symptoms, and the diagnosis of tuberculosis was made. She was finally examined by a member of this Society, who referred her to Dr. Jackson. The patient was referred back to Dr. Larkin, who treated her, and he told me yesterday that now, at the end of five years, the young lady is perfectly well and, so far as he can find, there is no evidence of recurrence.

I have seen another instance of recurrence which did not take place until four years after treatment by X-ray. One of the tumors showing atelectasis was treated elsewhere and the patient is now alive and well two years after the initial recognition.

Clinicians recognize the importance of early diagnosis, and we should realize that in the X-ray we have an instrument which will be beneficial in the treatment of these patients if there is early diagnosis. That is, after all, I think, the most important thing we can do for the patient. Malignancy is frightful wherever it is found; anything we can do to make the patient more comfortable should be done. We will arrive in some instances at an arrest of the case, and even, as in the instance of the first case mentioned,

at an apparent cure—an arrest for five years is certainly to be looked upon as desirable.

The diagnoses in these carcinomata of the bronchi were all made histologically, and, in some instances, by several different pathologists, so I have little doubt but that they were really of epithelial origin—true carcinomata.

The Results of Roentgen Therapy of Malignant Tumors in the Surgical Clinic of Göttingen, 1919-1927. Walter Schmidt. Strahlentherapie, 1928, XXX, 197.

Of a total of 523 cases of malignant tumor seen in the years from 1919 to 1927, 316 were alive at the time of the report, 217 had died, 71 (or 13 per cent) could not be followed up. [The arithmetic does not seem to be correct—*The Abstractor.*] The statistics cover carcinoma of the stomach, rectum, esophagus, pancreas, liver, skin, nose, eye, ear, lip, tongue, sinuses, tonsils, larynx, mediastinum, brain, bladder, prostate, penis, and breast, as well as sarcoma of the extremities, head, neck, or trunk. A study of the material leads the author to the following conclusions: The roentgen therapy of malignant operable tumors as examined in the surgical clinic can not compete with operation. The favorable results as observed in carcinoma of the cervix can not be duplicated in the realm of general surgery. Operable tumors should, therefore, go to surgery. An exception is the skin carcinoma, which may be treated by radiation if it is multiple or if the age of the patient or cosmetic reasons contra-indicate surgery. If there is no immediate response to radiation, radical removal should be done. Post-operative X-ray therapy is given in cases of breast carcinoma because the number of cures is definitely increased by that procedure. All cases of sarcoma are also treated following operation. All inoperable tumors, if they are not complicated by cachexia, are also subjected to radiation therapy. Very poor results are seen as a rule in carcinoma of the stomach. Local recurrence usually responds better than metastasis in lymph glands or bone. It is emphasized, however, that the analgesic effect in cases of bone metastasis is sometimes very striking.

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THE SIGNIFICANCE OF SMALL INTESTINAL STASIS[1]

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DURING our routine examinations of the gastro-intestinal tract at the University Hospital in Philadelphia, we have frequently been impressed by the unusual and varied appearances so commonly observed in the small intestine. We have often been at a loss to adequately interpret these apparent variations from the normal, and this, we dare say, has been the experience of most roentgenologists. The marked frequency of these atypical small intestinal forms has led us to undertake a study to determine, if possible, the cause of these apparent abnormalities. This presentation is a preliminary report of the problem and serves as an outline for future study. We hope that this subject will prove of sufficient interest to induce others to make similar observations.

Fig. 1. Showing the characteristics of the various portions of the small intestine: (*a*) Stippling of the proximal jejunum; (*b*) Serrations due to the valvulæ conniventes of the jejunum; (*c*) Terminal ileum.

Fig. 2. Normal distribution of the motor meal six hours after ingestion.

The small intestine is regarded by some authorities as the most essential portion of the alimentary canal. With this in mind one wonders why the diagnosis of small intestinal lesions has played so little part in the evolution of gastro-intestinal roentgenology. There are two reasons for this apparent

[1]Read before the Radiological Society of North America, at the Fourteenth Annual Meeting, at Chicago, Dec. 3–7, 1928.

Fig. 3. Ileal stasis. Only a small portion of the motor meal has reached the cecum. A case of chronic appendicitis, confirmed at operation.

Fig. 4. Small intestinal stasis due to post-operative adhesions.

neglect on the part of roentgenologists: First, in spite of its importance, the small intestine is not often the seat of an organic lesion; second, because of its size, mobility, and irregularity, the small intestine does not lend itself to roentgenologic study as favorably as the stomach or colon.

Recent observations, however, have led us to believe that much valuable information is to be obtained from a study of the small intestine. Physiologists and clinicians are becoming more and more impressed with the co-ordinated action of the various organs comprising the digestive system. Thus a disturbance of function in one organ reflects itself in a readjustment of function in the other organs of this system. No one is able to appreciate this fact better than the roentgenologist, who so frequently encounters disturbances in gastric function resulting from pathology elsewhere. The small intestine, situated as it is between the stomach and colon, must have its functions frequently altered by disturbances in either of these portions of the tract. Nature has wisely recognized this fact and has placed most of the small intestine upon a mesentery, thus permitting it to move about the abdomen with more or less freedom. Reflect for a moment on the many compromising situations that would arise were the jejunum and ileum fastened securely in place as is the duodenum.

One has but to think of the great variability of the symptomatology of diseases of the gastro-intestinal tract to conjecture that possibly, within the small bowel, lies the origin of many of those atypical symptoms which so frequently tax the diagnostic acumen of the physician. There are no diseases in the whole realm of medicine which so seldom run true to clinical types as do those of the gastro-intestinal tract. Clinical medicine has made but little more progress than has roentgenology in arriving at a true appreciation of small intestinal function. As in

Fig. 5. Small intestinal stasis due to a subacute peritonitis accompanying acute cholecystitis. Operative diagnosis.

Fig. 6. Small intestinal stasis accompanying acute diverticulitis.

other portions of the alimentary canal, we are of the belief that roentgenology offers a means whereby small intestinal function can be brought into its proper co-ordinated relationship to the function of other portions of the digestive tract.

In this study of the small intestine, only the jejunum and ileum are to be considered. The duodenum, both in its anatomy and physiology, differs so markedly from the rest of the small bowel as to be almost an entirely different organ. This is certainly true from a roentgenologic standpoint.

The most frequent manifestation of small intestinal dysfunction is that of stasis. This condition is probably more frequent than most roentgenologists appreciate. Mills (1), in 1922, gave some very interesting statistics on the subject. In the examination of 5,735 patients during a period of five years he found atypical small intestinal forms in 536 cases, or 9.3 per cent. During a six-month period, after having become particularly interested in the subject and after having gained considerable experience in recognizing the condition, he found 205 cases in 752 examinations, or 27.2 per cent. This marked increase in incidence clearly indicates the number of cases he must have overlooked in his larger series. Mills apparently was dealing with the more obvious cases of small intestinal stasis. Our own observations, which are in no manner complete, would indicate that about 50 per cent of our cases present some degree of small intestinal stasis.

It is needless to say that a clear understanding of normal small intestinal motility is a prerequisite to a proper appreciation of small intestinal stasis. Much work has been done by eminent physiologists and roentgenologists on normal motility. The outcome of this work has been to establish the fact that the average time required for the head of an opaque meal to traverse the entire small intestine is approximately 4½ hours.

Fig. 7. Small intestinal stasis resulting from ascites.

Fig. 8. Small intestinal stasis in ascites. Note the widely separated and gas-distended loops of small bowel.

At 6 hours the head of the column has reached the hepatic flexure, at which time a variable amount of the contents is still found in the terminal loops of the ileum. At 8 hours the small intestine should be empty. Variations constituting slight degrees of hypo- and hypermotility which are well within normal limits are frequently encountered.

The individual roentgenologist's interpretation of small intestinal stasis will depend in a large measure upon the technic employed in gastro-intestinal examinations. Our own work is done with the following technic. Six hours before the first examination the patient is given a motor meal consisting of 2 ounces of bismuth subcarbonate well stirred in a glass of buttermilk and taken with an ordinary breakfast. No previous laxatives are given unless the patient is in the habit of taking one every day. No food, of course, is permitted and as little water as possible is taken during the 6-hour period. With this technic, at the 6-hour examination we normally find the head of the column either in the middle of the ascending colon or at the hepatic flexure (Fig. 2). The terminal portion of the ileum is always filled. This usually appears as a mass of opaque material in the lower right quadrant of the abdomen. The patient at this time is given two glasses each containing 2 ounces of barium sulphate in buttermilk and fluoroscopic observations are made of the stomach and duodenum in the erect posture. A large film of the abdomen is then made in this position. Usually, individual loops of the ileum can not be distinguished in this film. The patient then assumes the recumbent posture and fluoroscopic examination is made of the stomach and duodenum in various positions. Further observations of the small intestine are made at this time. In addition to the contents in the terminal ileum

Fig. 9. Small intestinal stasis characteristic of tuberculous peritonitis. Operative confirmation.

Fig. 10. Small intestinal stasis resulting from an ulcer of the ileum. Diagnosis confirmed at operation.

a few scattered masses of opaque material are frequently seen in the small intestine. These never take the form of individual intestinal loops and apparently are of no significance. In the upper abdomen, contents are to be noted in the proximal jejunum. This is material from the second meal, is easily recognized as such, and, of course, is not likely to be mistaken for stasis of the motor meal. By palpation, the loops of the ileum can usually be separated. Particular attention is given to the ability to separate the terminal ileum from the cecum, the visualization of the appendix, and the detection of tenderness over the tip of the cecum.

In the interpretation of small intestinal stasis one must give due consideration to the emptying time of the stomach. It is generally believed that the stomach regulates the motility of the rest of the gastro-intestinal tract. Apparently, there is no compensatory mechanism whereby a delay in gastric evacuation is taken care of by an increase in intestinal motility. The reverse is probably true, because, according to Alvarez (2), intestinal peristalsis is dependent in a measure upon intra-intestinal pressure. Thus a failure to normally distend the small bowel by a delay of the contents in the stomach tends to inhibit normal peristaltic action. Therefore, a slowing-up of gastric motility means a slowing-up all along the line. Slight 6-hour gastric residues give rise to appearances in the small intestine which are well within the range of normal variations of motility. *Moderate or marked residues usually give an appearance which cannot be distinguished from certain types of true small intestinal stasis.*

Thus far in our study we have been able to distinguish between two main groups of cases showing small intestinal stasis (Table I).

Fig. 11. Small intestinal stasis in a case of multiple polyps of the small bowel. Diagnosis confirmed at operation.

TABLE I.—SMALL INTESTINAL STASIS

A. Ileal stasis
Due to lesions in the large bowel or terminal ileum.

B. Generalized small intestinal stasis
Due to
1. Extrinsic lesions producing mechanical obstruction
   (*a*) Adhesions
   (*b*) Subacute and chronic peritonitis
   (*c*) Ascites
2. Intrinsic lesions involving the lumen of the small intestine
   (*a*) Ulcers
   (*b*) Diverticula
   (*c*) Polyps
   (*d*) Primary neoplasms
3. Disturbances in the nervous mechanism
   (*a*) Splanchnic inhibition
   (*b*) Drugs

(A) The first is that group which has received considerable attention from roentgenologists and which is spoken of as "ileal stasis." Briefly, the characteristics of this group consist primarily in a delay in the advance of the head of the column of opaque material. This, at 6 hours, usually has extended only as far as the cecum (Fig. 3). Occasionally the entire meal is in the terminal ileum. Here we find the meal collected in one large mass in the lower right quadrant, with the individual intestinal loops usually not distinguishable. Palpation may serve to separate the loops, at which time we may note that they are of a somewhat larger caliber than normal. Frequently, the loops cannot be separated and often the terminal ileum is adherent to the side of the cecum. It is in this type of case particularly that the ileum so frequently is found deep in the pelvis. Placing the patient in the Trendelenburg position often fails to bring the bowel within reach of the palpating fingers.

The mechanism underlying the cause of this form of stasis apparently is well understood, since it is primarily the result of a mechanical obstacle to motility, usually resident in the large bowel. Some of the more common causes are colonic stasis, with or without constipation. This stasis may be functional in origin, as results from visceroptosis, or it may be due to obstructive lesions, such as adhesions, neoplasms of the large bowel, or external pressure from abdominal and pelvic tumors. Other important causes are congenital or acquired bands, membranes, and adhesions about the cecum and terminal ileum. Probably the most important single factor causing ileal stasis is adhesions resulting from inflammatory conditions of the appendix. This cause may be active whether there has been an appendectomy performed or not. Incompetency of the ileocecal sphincter, concerning which much has been written, is rarely if ever a primary cause of small intestinal stasis. In-

Fig. 12. Small intestinal stasis. The characteristic effect of morphine on intestinal motility in certain individuals. The patient had taken morphine for the relief of pain a short time prior to the roentgen examination.

Fig. 13. Same case as shown in Figure 12. No morphine taken. Re-examination for comparison with previous examination.

competency is usually secondary to one of the above enumerated conditions.

(*B*) The second group of cases showing small intestinal stasis differs markedly from the first. To begin with, the advancement of the head of the column is quite variable. While the entire motor meal may be in the small bowel, it is not unusual to find the head of the column at the splenic flexure or in the sigmoid colon. The most striking feature, however, is the atypical forms assumed by the intestinal shadows. One may find irregular filling of more or less discrete loops occupying various positions in the abdomen. The opaque material, while seen occasionally as one continuous shadow, is found more frequently as short segregated loops. Quite often the intestinal contents are broken up into masses which vary considerably in size and shape. The individual loops also show considerable variation in size, being occasionally of normal caliber but more frequently either contracted or distended. Quite as significant is the finding of gas-distended loops. The portion of the small bowel involved is indicated by the presence or absence of the serrations of the valvulæ conniventes of the jejunum. These latter loops, as a rule, will be found higher in the abdomen and more to the left. A frequent accompaniment of this condition is the detection of tenderness and irritability of the involved loops during fluoroscopic examination. This irritability is evidenced by regurgitation of contents, possibly due to antiperistalsis.

The causes of stasis in this group are quite as variable and numerous as in ileal stasis, but they can be classified under three headings: The first are external conditions producing a mechanical obstacle to intestinal motility; the second comprise those in-

Fig. 14. Small intestinal stasis. Roentgenologic diagnosis of multiple points of obstruction in the small bowel probably due to adhesions. At operation a duodenal ulcer with a localized peritonitis was found. Small intestines were entirely negative. Stasis in this instance thought to be due to splanchnic inhibition from localized peritonitis in splanchnic area.

Fig. 15. Small intestinal stasis due to splanchnic inhibition. At operation a large retroperitoneal tumor presenting in the splanchnic area was found.

trinsic lesions involving the lumen of the small intestine, and the third produce their effect by way of the nervous system.

1. Of the first group, namely, those extrinsic factors of a mechanical nature, the most common cause by far is adhesions (Fig. 4). Autopsy findings reveal that adhesions of the small intestine are quite common. These often result from surgical operations on the abdomen or from a previous peritonitis, this latter usually from some former attack of appendicitis. Another quite common cause of this form of stasis is an existing peritonitis, not the acute fulminating type accompanying a ruptured appendix, for these cases are seldom if ever subjected to a roentgenologic study, but one of the subacute and chronic varieties of peritonitis. Acute cholecystitis (Fig. 5) and penetrating peptic ulcers, accompanied by a subacute peritonitis, are associated quite commonly with small intestinal stasis. Of the same nature is the stasis accompanying acute diverticulitis (Fig. 6), to which attention has recently been called by Case and Upson (3). Under chronic peritonitis should be mentioned the stasis that practically always accompanies ascites (Fig. 7). The stasis in this condition is quite distinctive. The loops of bowel are discrete and widely separated from each other, being floated about in various parts of the abdomen by the free fluid present (Fig. 8). This fluid, however, interferes with the distinctness with which these loops are seen on the roentgenogram (Fig. 18). Probably the most classical example of this type of stasis is that seen in tuberculous peritonitis (Fig. 9). Here,

Fig. 16. Small intestinal stasis accompanying a carcinoma of the fundus of the stomach.

Fig. 17. Small intestinal stasis in a case of metastatic carcinoma of the liver, primary in the ovary.

small intestinal stasis is practically an invariable finding.

2. Probably least frequent among the causes of small intestinal stasis are primary lesions involving the lumen of the small bowel. These are quite commonly overlooked but occasionally one sees such lesions as ulcers (Fig. 10), diverticula, polyps (Fig. 11), and primary neoplasms. These are accompanied almost invariably by small intestinal stasis.

3. Intestinal stasis of nervous origin comprises a group of cases that bring us into the realm of speculation. The true nervous mechanism of small intestinal peristalsis is not fully understood. We know that the peristaltic activity of the small bowel is not dependent entirely upon its nerve supply, for when deprived of its nerves, rhythmic contractions occur which are an inherent property of the smooth muscle of the intestinal wall. Embedded in the intestinal muscles is the nerve plexus of Auerbach. According to Alvarez (2), it serves to conduct stimuli along the intestinal tract and to co-ordinate its movements. In addition to this plexus, we have the vagi and splanchnic nerves, which are said to have a regulatory influence upon peristalsis. In a general way, stimulation of the vagus tends to increase peristalsis, while the splanchnics exert an inhibitory action. It is readily seen that the influence of the nervous system upon intestinal motility is a very complex and confusing one, and thus it would be difficult to estimate how often *fear* during a roentgen examination is the cause of small intestinal stasis. Likewise, worry and illness, through the stimulation of the splanchnics, are undoubtedly responsible for some cases of stasis.

One of the best examples of small intestinal stasis due to nerve influence is the effect of morphine (Figs. 12 and 13), described some years ago by Pancoast and Hopkins (4). We have observed a somewhat simi-

Fig. 18. Small intestinal stasis in a case of carcinoma of the pylorus. Patient also had ascites. Note the wide separation of the intestinal loops and the indistinct outlines due to the presence of fluid.

lar but much less definite and characteristic effect from luminal.

There is ample experimental proof for the belief in reflex inhibition of small intestinal motility. This reflex is by way of the splanchnic nerves. It is thought that any type of peritoneal irritation will cause a reflex splanchnic inhibition of peristaltic activity. The well-known post-operative depression of intestinal peristalsis is probably an example of this kind. We have noted that lesions in the upper abdomen frequently are accompanied by small intestinal stasis. This has been seen in gall-bladder disease and in gastric and duodenal ulcers (Fig. 14), where at operation a local peritonitis or extensive adhesions involving the splanchnic area was found. Such involvement, we believe, produces an irritation of the splanchnic nerves and thus reflexly results in small intestinal stasis. Also, tumors of various kinds, especially retroperitoneal tumors (Fig. 15), presenting in the splanchnic area, have a similar effect on motility. Finally, it is our impression that small intestinal stasis is more common in elderly individuals—a manifestation of atony, resulting from loss of intestinal tone.

There is one cause of small intestinal sta-

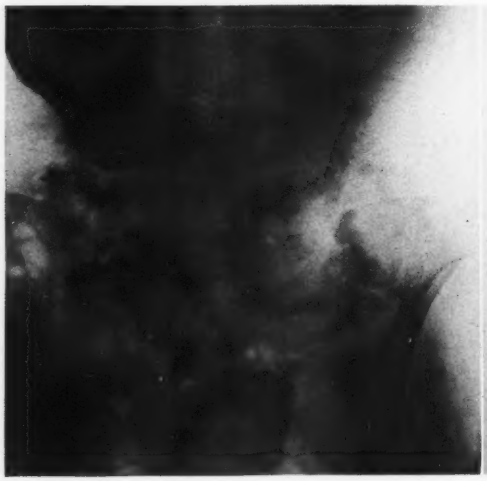

Fig. 19. Small intestinal stasis in a patient with a carcinoma of the tongue.

Fig. 20. Small intestinal stasis. Patient has a carcinoma of the breast, with metastasis to the pelvis. Stasis is probably the result of metastatic involvement of the mesenteric nodes.

sis, the significance of which we wish particularly to emphasize. This is the stasis so commonly associated with malignant disease. During the past year we have been particularly impressed by the frequency of this occurrence. The stasis in this instance is usually quite obvious and marked (Figs. 16 and 17). It assumes the characteristics found in stasis resulting from extensive adhesions. The cause of this form of stasis may be any one of various factors. Where there has been metastasis to the peritoneum, resulting in a chronic peritonitis, small intestinal stasis follows as a natural consequence. So extensive and adhesive may the peritonitis be under such circumstances that in rare instances the small bowel is matted together into a ball-like mass that can be felt through the abdominal wall. Peritoneal metastasis occurs most frequently in carcinomas of the stomach and ovaries and it is in these malignancies that small intestinal stasis is most commonly observed. Many ulcerative and infected carcinomas of the stomach and colon commonly produce a low-grade peritonitis of a non-malignant nature (Fig. 18). In such cases, also, stasis can be demonstrated. Metastatic involvement of the small intestine alone is not a common occurrence. Metastasis to the mesenteric nodes, however, occurs much more frequently. We believe that such involvement can give rise to small intestinal stasis reflexly by way of the splanchnic nerves. In addition to abdominal malignancies, we have seen small intestinal stasis in association with carcinoma of the tongue (Fig. 19) and breast (Fig. 20). In the extra-abdominal malignancies, stasis probably is due to metastasis to the mesenteric nodes. Alvarez (5) has noted diminution in peristaltic activity in animals that have become debilitated from one cause or another. This may account for the small intestinal stasis observed in malignant patients in whom emaciation has become a prominent feature.

Fig. 21. Small intestinal stasis. Not characteristic on roentgenogram. Diagnosed by fluoroscopic observation of irritability of and regurgitation in the small bowel. Roentgen examination otherwise negative. On basis of stasis, malignancy suspected. Barium enema recommended, but refused. Patient later developed acute intestinal obstruction. Surgeon reported carcinoma at rectosigmoid junction.

We have come to regard this roentgenologic sign of malignancy as of inestimable value. A clinical suspicion of malignancy warns us to pay particular attention to the small bowel, and a finding of small intestinal stasis has caused us to suspect and search for malignancy (Fig. 21), sometimes with fruitful results. Small intestinal stasis, therefore, occurring in a patient of cancer age, gains more significance and should lead to a thorough study of the patient to determine, if possible, its exact cause.

The few observations made thus far have led us to attach considerable importance to the finding of small intestinal stasis. While it is not possible in every case to assign a definite cause for this phenomenon, we believe that further experience will enable us to explain many of those cases that now go

unaccounted for. We also believe that such observations will give us a better insight into the actual pathology existing in the peritoneal cavity and a truer appreciation of its influence upon gastro-intestinal function.

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DISCUSSION

Dr. H. K. Pancoast (Philadelphia): My interest in the small intestinal stasis was aroused a number of years ago when we had a case presenting the exact appearance Dr. Kornblum has shown in the instance in which morphine had been given the night before for pain. I showed these old plates at a meeting and Dr. Hickey suggested that I look up the literature and write up the subject. Dr. Arthur Hopkins and I then instituted an investigation with the use of various drugs. The effect of morphine was the most pronounced. In some, stasis in the small intestine was produced, while in others the effect was largely upon the stomach. In the case Dr. Kornblum showed, when the surgeon came to see the films the next day I asked him how much morphine this patient had had, and he asked me how I knew the patient had any morphine. I said: "The appearance of the small bowel is so characteristic that no one could be misled by it."

The late Dr. Walter Mills made many contributions to roentgenology by his investigations on the small intestinal tract, and, as in many other investigations of his, we lost much through his death. There are many instances in which the study of the small intestine may be of great value. In a case of gall-bladder disease with active symptoms, or of an ulcer, if you find small intestinal stasis along with the other findings, you must at least suspect that there may be some leakage or some peritoneal irritation. In one of our cases with stasis a sticky peritoneum was found at operation in connection with an acute gall bladder.

In small intestine stasis, one may find many loops of small intestines lying idle (not blocked but simply lying idle), whereas normally the contents pass through the duodenum and jejunum rather rapidly. In such cases one must always suspect or look for some cause for this appearance. In other cases there may be stasis at isolated points, and one will see back-and-forth movements of the contents, which will be repeated time and again. In such cases one must always suspect trouble. An actual block or obstruction in the small intestine presents an entirely different appearance.

One must be on guard against a diagnosis of chronic appendicitis on the basis of ileal stasis, because the appearance of stasis may be due to such a large number of causes. As a matter of fact it has been many years since we have made a diagnosis of chronic appendicitis by roentgen-ray examination. We report ileal stasis for what it may be worth. Undoubted tenderness over the appendix is reported for what it may be worth, but the only finding we give any credence to is the absolute demonstration of adhesions of the appendix.

THE INCIPIENCY OF DISEASE[1]

By W. WALTER WASSON. M.D., DENVER, COLORADO

IN the evolutionary course of the practice of medicine one finds the general practitioner or the specialist who thinks in terms of adult pathology and the pediatrician who thinks in terms of child pathology. The latter represents a group of practitioners that has evolved as a result of the difficulty of unravelling some of the disorders of infancy and childhood. The infant or child is unable to describe his symptoms and there has been no way of disclosing the pathology of the internal organs other than by the senses of sight, touch, and hearing of those concerned in his care. The diseases of this period have, as a result, been obscure, and the treatment has, in the great part, resolved itself into a question of diet and general hygiene. On the other hand, those dealing with adult pathology have been more fortunate, and great discoveries have been made, in methods of both diagnosis and treatment. While this latter group of physicians will occasionally speak of a period of "defective health" preceding the onset of manifest disease, there seems to have been no great effort put forth in connecting the manifest diseases of adult life with the obscure conditions of infancy and childhood. In fact, it has been taught that the infant or child will outgrow his ailments, and that such conditions as sinusitis do not occur in infancy. Even in our medical schools there has been no correlation between these two extremes of life, and it is the unusual medical student who can describe the development of the nasal accessory sinuses from birth to adult life.

In a survey of the medical literature, tuberculosis is the outstanding exception to the situation just described, but even in this extensively studied subject there is a great gap between the earliest known infection of Ghon (1), occurring in infancy, and the clinical tuberculosis of adult life. Much attention is paid in the literature to the care of the child on general principles, assuming that whatever is done for the hygienic conditions of the infant and child will be of material aid, yet very little is taught as to specific conditions. Arteriosclerosis is described as the disease typical of senility, and asthma, sinus infection, appendicitis, lesions of the urinary tract, and in fact almost all chronic diseases, are thought to be peculiar to individuals past forty. As a result of such teaching the man of fifty is seeking the "Fountain of Youth."

In 1918 (2) I presented a paper before this Society and in this city on the short exposure technic in the radiography of the chest. This short exposure technic made it possible to portray upon the radiographic film not only the anatomy of the adult, with its physiological and pathological variations, but for the first time that of the true situation with regard to the infant. When the interpretations of such radiographs are preceded by the necessary fundamental studies the radiographs then become of value often greater than an autopsy, as they record events both preceding and following a certain moment or period of time. It became possible to record the life span of any individual from birth by means of the radiograph, as well as by other methods of examination. After ten years of such study there has evolved a concept of disease different in many respects from that usually taught in medicine. There necessarily has accumulated a mass of evidence, since many of our children have been studied from birth to seven years of age. As much of this material has been presented in other papers

[1]Presented before the Fourteenth Annual Meeting of the Radiological Society of North America, in Chicago, December 3–7, 1928. From the Child Research Council, University of Colorado School of Medicine, Denver, Colorado.

(3) no special attempt will be made to restate it in this paper. After this lapse of time it does not seem too early to make this report and describe the trend of our investigations.

Disease is a relative term and in each one's mind it denotes a degree of pathology according to that individual's experience. We all admit that it is a condition of disordered physiology, but seldom can the change in physiology be detected at its very beginning. However, for my purpose I do not wish to consider disease as confined to that condition of pathology which gives clinical evidence, since one's ability to obtain such clinical evidence concerning a patient in early infancy and childhood is inadequate—a result of our lack of knowledge rather than that such pathology does not exist. I do not wish to be concerned with etiology, describing certain infections or injuries as the cause of certain diseases, nor with the prevalence of the various diseases. Rather, I wish to discuss the time in the individual's life span when certain diseases have their incipiency and at which period they can be detected and placed according to their entity if we have but studied the human being with these thoughts in mind. No doubt the changes in the cell membrane or the chromosomes or the dysfunction of the tissue unit of Gruner (4) are the earliest beginnings of disease and make possible the various infections or the disorders in metabolism, but such discussion I must leave to the histologist or the biochemist. It would seem appropriate to ask ourselves the question as to what time in life any particular process of pathology may be detected and diagnosed if all the methods are used which are now available. It would seem to me that an effort to answer this question should be our first undertaking, and a very important one. The result of such an investigation might take us back forty years in the life of a man to a time when all of our present knowledge of that period of his life was not only vague as to scientific information but likewise impossible for him to recall. Not being recorded and not being understood, the events of the early period of life are not connected with any condition forty years later. Such an investigation might give us a different conception of the pathology as existing in this man of fifty, and likewise a different conception of what our treatment should be. An end-result, following upon many decades of life, is difficult to overcome, either medically or surgically, with any hope of a complete restoration to health.

There has been much evidence presented that certain diseases have their beginning in the anatomical variations transmitted by heredity—Geddes (5) has laid much emphasis upon these factors. Individuals have like variations in their internal organs as in their external characteristics. Radiologists often remember patients by the variations of their internal organs as revealed by the radiograph. The body fluids have certain characteristics, such as the quality of resistance to disease, transmitted, no doubt, by heredity. Certain pathologic conditions, as pneumonia (6), have been discovered in infants at birth, but, of all diseases, syphilis is the one outstanding example transmittible from parent to embryo. In spite of these facts, the thing of greatest importance is that we inherit our environment.

Upon the birth of the infant with its inherited background, it is immediately brought into contact with certain great factors of the physical world. It is immediately forced to meet a complex of exposures, trauma, food conditions, nerve stresses, and infections. It has been our teaching in the past that very little infection of the infant takes place in the first few months of life, other than an occasional pneumonia or some of the eruptive fevers. If the usual infections of adult life can be proven to exist in the first few months of life, it certainly

will alter our entire concept of disease. It is illogical to assume that the individual whose body is composed of many systems, and each system, in turn, composed of many organs, will reach the age of twenty absolutely free from disease. It has been our assumption that the natural tendency to health and vigor would carry him through this period. Is such an assumption—that the processes of life at this period are so entirely directed towards the establishment and maintenance of normal physiology—correct? Certainly in the physical world about us this is not true. Here it is impossible for any body of matter to be created that many physical factors will not immediately begin to act towards its destruction and will ultimately destroy it. Why should the human being be any exception to this rule? Perhaps the inability of the infant to talk covers over a progressive pathology. Scammon (7) has recognized this possibility of progression, as applied to anatomy, in his endeavor to fill in the gaps in anatomy between the infant and the adult. It would seem that we should consider pathology from the same standpoint of progression, rather than jump too suddenly from histology to the pathology of manifest diseases. As stated before, while arteriosclerosis is considered a disease typical of senility, yet Aschoff (8) has described atheromatous spots on the valves of the heart and in the aorta, both in the age-periods of infancy and puberty, and has described them as marking the earlier stages of senile arteriosclerosis. I have repeatedly observed sclerotic changes in arteries, especially the iliacs, of boys in their early twenties.

Various urologists (9) have stated that they find, with a few exceptions, the same lesions of the urinary tract in the infant and child as in the adult. Smith (9) concludes: "Accumulating experience indicates there is no essential difference between the urologic problems encountered in children and in adults, and that calculi occur in approximately the same ratio as in adults." In the examination of the school children of to-day, observers report every known disease and in considerable prevalence. The psychiatrists observe many mental and nervous disorders at an early age.

Turning now to the investigations of the Child Research Council at the University of Colorado School of Medicine, it is impossible at this time to present all the data that have accumulated or describe all of the various phases, but certain examples may be cited to illustrate the points under discussion. In the beginning I may say that the outstanding observation in the study of a number of children from birth, children who have had definite periodic examinations from the time of birth, is the sequence of events. This sequence is very striking and gives an entirely different concept from that obtained by a study of groups. In group studies, since the individuals have not been seen before and are not followed after the examination, it is difficult to connect one group with another and visualize in one's mind the real sequence, either of physiologic or pathologic conditions. As the respiratory infections are the most prominent diseases in the first decade of life, I will cite various examples of these infections.

Dr. Carmody's (10) studies, together with radiographic examinations, have definitely demonstrated that not only does sinus infection occur in the first few weeks or months of life, but that it is quite prevalent. Many such cases have been watched over a period of years, observing not only the development of the sinuses but the course of this infection, with its periodic exacerbations and remissions. Many of these cases have refused treatment, a circumstance which has enabled us to observe and record the influence of this sinus infection upon the hila of the lungs, observing the changes

from the normal hilum of the infant of a few weeks of age to the definitely diseased one of a few years of age. I have described certain of these changes at the hila to be typical of bronchosinusitis disease. Cunningham (11) has found this condition of the sinuses and hila to be a basis for bronchial asthma in infants and has reported several such cases at a year or fifteen months of age. I have reported this same condition as a basis for so-called thymic stridor (12) in infants a few weeks of age, and this stridor appears to be a forerunner of either true bronchitis or bronchial asthma, as described by Cunningham. It is observed by such studies that at this period of life the sinuses begin to take on the characteristics to be seen in adults, not only as to size and shape, but as to clearness or densities, indicating an infection as revealed by the radiograph. The pathology as laid down in the hilum of the lung, just mentioned, also resembles that of the adult asthmatic or bronchitic. One of our cases suffering with bronchosinusitis disease and the symptoms of bronchial asthma at three years of age possesses the symptoms not only of his father, who suffers with hay-fever and bronchial asthma, but of the grandfather and great-grandfather. And in each case of the parentage, the history reveals that the condition began in childhood. In every case of our series in which there is marked sinus infection, with bronchitis or bronchiectasis in the parent, the infant shows the same condition. These observations, just mentioned, have been discussed in detail in a previous paper on the Hilus of the Lung (13). In our studies of those children infected with tuberculosis, it is observed that the markings on the radiograph, so often spoken of as typical of the adult chest, are laid down before the infant's fifth year. Here again, the sequence of events from the earliest tuberculous infection of the infant through the early years of life is instructive.

In other words, the investigations of the Child Research Council have proven to me that the infections of the respiratory tract, such as one finds in the adult, do actually occur in the infant and young child, and that the resulting pathology, as laid down by these infections, is similar to that found in the adult. This pathology is, however, of a purer form, uncomplicated by years of life's disasters and much easier of study. Furthermore, these infections are much more prevalent than the medical profession is taught, and their importance is difficult to overestimate. They certainly are the most important diseases of the first decade of life. Their exacerbations and remissions, extending over a period of years, have not been understood and their treatment has been woefully neglected.

Let us now turn to a consideration of adults as we find them in private practice. If this concept of the early beginning of disease is applied to our private cases, one is surprised to observe how often the intelligent adult patient, suffering with bronchitis, asthma, sinus infection, and the various other chronic diseases, will state, upon close questioning, that he has had "more or less" of this condition as long as he can remember. Furthermore, I have had the opportunity to study a large group of children of all ages at the Children's Hospital of Denver and find that the above observations, as to type and duration of disease, are further borne out. Not only does tuberculosis reveal itself in certain families, but likewise sinus infection, bronchitis, and bronchial asthma are more prevalent among those children whose parents are suffering with these diseases.

SUMMARY

In summarizing, I would present certain facts to be considered when one thinks of the incipiency of disease:

1. Hereditary conditions, including both anatomical variations and environment.

2. The great factor of combat to be encountered by the individual immediately upon his birth.

3. Infection of many types does actually take place as early as the first few weeks of life.

4. The pathology laid down by this infection is similar to the pathology found later in adults and is progressive in character.

5. Histories of intelligent adult patients date the beginning of their chronic ailments back to childhood.

After due consideration of these facts, especially when one has had the opportunity to observe the sequence of events in a child's life from birth to a few years of age, one must conclude that almost all chronic diseases are steadily progressive conditions, with their incipiency in infancy or childhood and their end-results in adult life. In such a progress of disease from birth to old age, one must be careful not to take any particular stage as a standard or guide. Upon these chronic disorders of the body are implanted the acute diseases, and it is often difficult to say whether any particular condition is an acute and different infection or an exacerbation of the pre-existing one. Such a concept of placing the progression of disease foremost in one's thoughts is very important both for diagnosis and treatment. It stimulates us to think of the beginning of disease, its progress during the span of life of our patient, its exacerbations and remissions, the influence of proper habits and living conditions upon its progress, and, finally, not only how much pathology actually exists at the time of our examination, but how much is our evaluation of this pathology to be influenced by pre-existing conditions of life. I am aware that many of the diseases of infancy are being overlooked, while the great opportunity for prevention is allowed to pass. It is the radiograph which has made such studies possible and yet the interest in this field has not yet begun. The radiologist and internist are continually striving to state the age of any lesion, as so often found in the chest or sinuses, but their knowledge of the pre-existing pathology has been inadequate. A retrocecal appendix which has existed from birth is not necessarily of itself an indication for removal, but, rather, one should ask the question, What pathology has taken place in this appendix during the previous forty years, and is it materially affecting the health of the patient?

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DISCUSSION

DR. W. W. WATKINS (Phoenix, Arizona): This paper, with a rather prophetic tone in its discussion, is a little difficult for one who had not previously seen the paper, but has just heard it, to discuss. We know how our specialty has applied its mechanics and its development to the elucidation of disease already developed. We now have a new outlook presented to us and probably will find ourselves studying disease in its development. The probabilities are that ten years hence we will, in looking back upon our work, find ourselves very much in the position of the Presbyterian synod which some years ago was discussing the subject of infant damnation, and a resolution was introduced, "Resolved, that infants will not suffer damnation if they are not baptized before death." Some one in the audience arose and wanted to know about the resolution; he said it seemed all right but he wished to know "what was going to happen to all those babies who died before this resolution was passed." We probably will find ourselves wondering ten years from now how we have gotten along with diseases of babies and children without this work, presenting to us the application of the science of radiology to the study of the early stages of disease, particularly in its recent development. Dr. Wasson has been one of the pioneers in this work and a very earnest and indefatigable worker in this new field.

The development of radiology along this new line has resulted from two causes: one has been the improvement in technic, so that earlier evidences of tissue change could be detected; of more importance has been the apotheosis of the roentgenologist from a somewhat chaotic mixture of clinician, pathologist, and technician. The radiologist must be all three and yet be an entity in himself. From the older conception, that X-ray methods could show only well-developed stages of pathologic changes, we are rapidly developing methods of detecting tissue changes which would otherwise remain absolutely unsuspected. We are detecting disease before disease is clinically present. In the chest the clinician now expects us to show the X-ray evidence of the premier complex, where the first battle between bacilli and tissue cells was fought and won by the tissues. Formerly we were quite well pleased if we could show gallstones—the final stage in the development of gallbladder disease. Now, we are expected to show, by functional dye tests, the evidences of disease before the palpating fingers of the surgeon can feel them. In sinus disease, we must show by changes in tissue density, the evidences of disease at a stage when lavage of the antrum is still negative. And so it goes throughout the body. We can move confidently into this relatively new field of incipient disease only if we remember always that radiology is a highly developed segment of clinical medicine, that it must rest upon a tripod of clinical knowledge, pathologic knowledge, and technical skill. As for every specialty in medicine, the first and prime requisite is a knowledge of clinical medicine; to its problems we can then apply our special training in radiology. And to my mind this is one of the important lessons being developed from Dr. Wasson's work in the early stages of disease.

DR. W. W. WASSON (closing): I showed no slides, as I have previously shown slides before this Society. There are a few films in the Scientific Exhibit

illustrating at least the sinus side of this situation. I brought no chest films along with them, as I did not wish to complicate the exhibit.

In answer to Dr. Clement's question, we have studied the lung from the time of birth, watching the appearance of infection, watching its progress, trying to correlate the influences of the sinuses upon the chest, watching the early stages of tuberculosis. It is a very interesting subject, at least to me, and I think it will be to others of you as you become better acquainted with it. I believe that the pathology is pure in infancy. Frequently, I think, we have some tuberculosis without other complications. We have seen the influence of sinus infection on the chest without tuberculosis. We have seen the two conditions upon the same radiograph.

Now, of course, we ask the questions: What is the influence of this mixed infection upon tuberculosis in the early infant? Will these children with pure tuberculosis do better than children with tuberculosis and sinus infection? There are many fields of investigation of this type that will occur to us, but the important one for us to study in the beginning is the ability to differentiate upon the radiographs between the sinus infection and its influence upon the hilus of the lungs and that of tuberculosis. There are radiologists who will be able to do this. Then will come the mixture of the two conditions. These are more difficult. But if we can recognize tuberculosis in the infant as distinguished from sinus infection and its influence, then we will be better able to differentiate between the two when seen on the same radiograph. I am thoroughly convinced that we are overlooking many things in the young child which are of very great importance to his later life.

THE PRESENT STATUS OF THE CANCER PROBLEM[1]

By GEORGE A. SOPER, PH.D., NEW YORK

WHAT do we mean by the "cancer problem," what is the world doing to solve it, and how far have we advanced toward the accomplishment of this task? These are the questions which I propose to touch upon.

In attempting to answer these questions it will be necessary to take account of our common knowledge of the subject, and, at times, to repeat what you already well know. My hope is that, out of the collection of facts and opinions to which I can but briefly refer, a mosaic may be made of the problem which cancer presents and of the need of increasing our efforts to solve it.

On the basis of the mortality statistics issued by the United States Census Bureau it is estimated that over 113,000 persons die of cancer in the United States every year and it is believed that the death rate is increasing. The official statistics are not accurate in showing the prevalence of this disease; it is known that the data for cancer give an inadequate idea of the mortality.

But, taking the United States statistics as they are, it appears that more people die of cancer than of all the infectious diseases combined, excepting tuberculosis. More people die of cancer than are killed by railroads, street cars, automobiles, fire, drowning, mining accidents, machinery, poisons, homicides and suicides, all put together. According to the official statistics, one in ten of the men and women now living in this country is destined to die of cancer, and of all women who die between the ages of 45 and 65 years, one out of five will succumb to it.

Practically every case is fatal unless it is treated skillfully and in its early stages, or unless the patient falls a victim to some other disease before the cancer can run its course. Cancer is, in fact, the most fatal disease known to modern civilization and one of the most prevalent. It is probable that the number of persons with cancer in the United States exceeds 400,000.

How to eradicate cancer, or, at least, to subjugate it to such an extent that persons using reasonable care, and medical men and public health authorities employing practicable measures against it, will be capable of reducing its prevalence to that of the minor infectious diseases, let us say, constitutes the cancer problem.

According to the opinion of eminent pathologists, what is termed "cancer" is not really a single disease, as is commonly supposed, but a group of diseases. If this is so, and they can be separated and distinguished one from another, the statistical and epidemiological study of cancer will be greatly facilitated.

Investigations of breast cancer and uterine cancer which have been made on a large scale by the Health Committee of the League of Nations show that marked differences in the mortality occur among people of different countries, but the reasons for these differences have not been explained. Differences sometimes occur in different parts of the same country and among definite groups in a single city without any obvious reason.

Here lies a field of investigation which needs to be cultivated, for it is to be noted that cancer is not, apparently, contagious or infectious nor due to a specific parasite of any kind. No article or condition of food, water, air, clothing, or personal habit has ever been proved to be connected with it in a causative way.

[1]Read before the Radiological Society of North America, at the Fourteenth Annual Meeting, at Chicago, Dec. 3–7, 1928.

Usually, but not always, some form of chronic irritation precedes the growth. Sometimes months, and even years, after the irritation has ceased the cancer makes its appearance. Sometimes a cancer develops in a person where no detectable irritation has occurred. Frequently it does not affect places where long continued irritations have taken place. To say that cancer is always the result of irritation, therefore, requires that a new and unusual definition be given to that word.

The preponderance of opinion is that cancer is not a constitutional disease with a local manifestation, but a local condition at first. Something goes wrong with the regulating mechanism which controls the normal behavior of the cells of which the body is composed. They mutiny, as it were, against their government, which is then overturned, and enter upon a career of unrestrained and unrestrainable multiplication. This multiplication is a characteristic of all cancers. Usually the resulting growth bears a more or less close resemblance to the part from which it sprang, but is markedly inferior to it. The individual cells and communal arrangements such as blood vessels and nerves are deficient or absent. The outlaw community cannot properly defend itself, and, if injured, becomes a ready prey to bacterial infection, with its attendant destruction and foul odors.

A cancer usually destroys its victim's life by interfering with the organs and other parts where it occurs, and through the separation of small particles, which are carried to other parts of the body, where new cancers develop. When this migration takes place, the patient is usually beyond cure, for these new centers of growth cannot all be reached by any procedure which has thus far been devised.

Here, I may say parenthetically, is one of the great weaknesses in our method of attacking cancer. With our present knowledge, only cancers in certain favorable locations and in certain favorable stages are susceptible of cure, and it may be said candidly and without circumlocution that cure always means removal or destruction of the offending material.

According to the International Cancer Congress held in America in 1926, cancer is not itself hereditary, although a certain susceptibility or predisposition seems to exist in some families for a few generations at least. This susceptibility has sometimes a relation to a particular organ. It is, perhaps, cancer of the breast, which repeatedly occurs in a family.

Among causative factors, conditions associated with certain industrial occupations are believed occasionally to play a part. Much has been written about the cancers of chimney sweeps, mule spinners, and anilin workers. It cannot be said, however, that the existing knowledge of the subject warrants discrimination against any industrial occupation, except in very unusual circumstances.

A certain amount of preventive doctrine has been built up about the irritation theory of the causation of cancer. Thus, people are cautioned to have their dental work promptly attended to, lest some irritating point lead to cancer of the mouth. We are told that moles and certain other skin imperfections must not be continually rubbed or otherwise irritated, or cancers may develop at these points. Dark, pigmented moles should be removed or watched for any change in their size or appearance, particularly after the age of 30 years, for they sometimes lead to the most rapid and fatal forms of cancer. Lacerations of the cervix produced by childbirth should be repaired. These are examples of the precautions which are urged against the production of cancers in the various parts of the body. Insofar as they are effective and of general application, they help to make cancer a preventable disease.

There are no sanitary or other general procedures which apply to the prevention of

cancer, such as are available for the prevention of many other diseases, but in the hygiene of the mouth, generative organs and some other parts of the body there is a promising field for preventive effort.

All of the resources which are now available for a war against cancer are medical, or, rather, surgical and radiological. They require the co-operation of the patient. They are efficient in proportion to (1) the discoverability of the cancer, (2) the location of the cancer, (3) the stage of advancement of the cancer, and (4) the equipment, skill, and ability which are available to diagnose and treat the case.

To effect a cure the case must be discovered early and treated skillfully, and it is obvious that the cancer must be so located as to admit of this.

It is essential that the first treatment shall be the right treatment. One of the lamentable aspects of the problem lies in the large proportion of patients with advanced and incurable cancer who apply at hospitals, having had one improper method of treatment after another tried upon them. It is encouraging to learn that patients are coming earlier to physicians and hospitals than they formerly did, but the proportion which these bear to the whole is still very small.

People often ask how great a saving in lives and suffering could be accomplished if all the information which now exists concerning the prevention and cure of the disease were generally employed. Some have put the lives that might be saved at one-half the deaths which now occur among women and one-third of those among men, while others have placed the possible saving much lower. There are no reliable statistics to go by: it is a matter of opinion. The question is an academic one, for the reason that the useful knowledge about cancer is not, and cannot be, universally distributed and employed. It is more practical to consider the saving which is probably effected at the present time.

A study of the average treatment of cancer in nineteen American cities recently made by Dr. Saltzstein, of Detroit, and published in the *Journal of the American Medical Association,* shows that, excluding medical centers where exceptional skill is available, the potentially curable cancer, aside from cancer of the skin, is cancer of the breast. This is a dark picture. Cancers of the breast constitute about 9 per cent of all cancers and cancers of the skin less than 3 per cent. Cancers of the female genital organs (15 per cent) and cancers of the gastro-intestinal tract (about 50 per cent) are commonly so far advanced as to be hopeless when the patients reach those who are skillful in treating such cancers in their early stages. To say that 1 per cent of all cancer cases are now getting into the hands of those who are competent to cure them while there is still time to make a cure possible would be an exaggeration. This situation can and must be improved.

The number of cures possible depends partly upon what is meant by the word "cure." The usual meaning of this word does not apply, because cancers sometimes recur long after all signs of them have been removed. The attempt to avoid this difficulty by speaking of "three-year cures" or "five-year cures" does not wholly meet the situation. Longer periods should be taken through which the patient must be free from a recurrence. To be actually cured a patient should remain free from all growths attributable to the cancer for the rest of his life. In this matter we should, I think, be guided by our Swedish brothers, who have done so much to put the treatment of cancer by irradiation upon a scientific basis, and follow up the case to the very end.

Research is being carried on in practically every civilized country, looking to the discovery of the cause or causes of cancer: there is hardly a conceivable direction in which such studies are not being made.

Some of this work is being done in institutes especially established for the purpose. Usually there are highly trained investigators at the head of groups of skilled assistants, constituting scientific units. Their methods and results are published in journals which have a limited circulation, for there are few who can, or care to, follow them. It is estimated that, all told, about half a million dollars a year is being spent in Europe and America in this way.

A good deal of laboratory research (for the most part, with inadequate facilities) is being done by individuals working alone or with such help as they can find a way for which to pay, not a few duplicating work which has been done already by others. The laboratory investigation of the major questions of cancer to-day requires an amount of training, equipment, and personal help which entitles this calling to rank as a special profession.

The usual lines of investigation into the causation of cancer assume that cancer is due to an extrinsic factor or to an intrinsic factor or to a combination of both.

The microbic theory is not dead—a few of the best known workers still hold to it. Then there are advocates of the idea that an ultra-microscopic virus, combined with an activating substance, is necessary and sufficient to cause cancer. Finally, there are those who look to spontaneous changes in the physiology of the cells to account for the condition. The opinion most frequently met with is that cancer may be produced in a number of ways and that it is a train of events acting upon a susceptible person rather than a single causative factor which leads to the result. Obviously, any break in the chain or increase in resistance would afford protection.

Animal experimentation, which has yielded much information, continues to occupy a foremost place in laboratory work, and tissue culture, affording as it does opportunities to study the physiology of the normal and cancer cell, is also a favorite method of research.

Epidemiological research is being prosecuted vigorously. It received much attention at the International Cancer Conference at London last Summer [1928]. Unquestionably it is capable of yielding valuable results in the future, as it has done in the past, providing persons of exceptional qualifications can be found to engage in it.

It is a far cry from scientific research of the kinds referred to to the solution of the cancer problem by inexperienced laymen, and yet there have been those who have felt that the importance of the subject was so great that everybody should be encouraged to help solve it. An effort was made by a public-spirited citizen two years ago to stimulate inventive genius through the offer of a money reward for the discovery of an effective cure or a general and effective method of prevention. Fifty thousand dollars was offered as a prize for the accomplishment of either of these objects. The offer held open for one year, during which time over thirty-five hundred applications were made, practically all of which were worthless. It is the intention to publish a report upon this subject to show why none of the propositions submitted fulfilled the requirements, or could be expected to do so, in view of the existing scientific knowledge of cancer.

The financial resources which are available in the United States to support cancer research are entirely inadequate and should be greatly increased. In England a society has been formed to collect money from the public and apply it to the support of promising undertakings in this field, the society numbering among its members many of the most prominent medical men, scientists, and public health authorities in the country. One of the helpful things done by this organization is the publication of an index of the current literature of laboratory research.

Clinical research is making progress. Few

persons realize how great has been the accumulation of knowledge in this field during the last five years. Methods of diagnosis and treatment have both been improved.

The value of surgery is unquestioned, and, in some forms of cancer, standard procedures exist for the operations required. In the opinion of those who are in a position to know, it is doubtful whether the surgical technic can be more highly developed. The great question to-day is not how to employ surgery or irradiation, but how to get the patients who need this help to go in time to those who are qualified to give it. Too often the cancer gives no sign of its presence until too late for a cure to be effected. Too often the patient fails to attach proper significance to his condition until too late. Too often the patient is unable to find the medical help which he requires.

Thanks to the developments of radiology, the diagnosis of internal cancer has been placed upon a far more satisfactory basis than ever before, but here again people must apply for help in order to get it. There is no well-equipped general hospital to-day without its radiological department.

Radium and X-rays are coming to be recognized as of great and increasing value in the treatment of cancers, as their mastery through scientific and skillful application is being accomplished. At the International Cancer Conference to which reference has previously been made, the results which were shown to have been obtained in treating cancer in certain locations by irradiation were acknowledged to be quite as good as those produced by surgery, and without the attendant operative mortality. In some forms of cancer, irradiation by radium or X-rays is to be preferred to any other treatment.

This does not mean that anyone with a little radium is competent to treat cancer. Far from it. To treat cancer successfully by radium or X-rays requires a high degree of skill, and, to acquire this, special training and experience are indispensable.

The total quantity of radium in the United States to-day is probably about 75 grams—not nearly enough. A large hospital especially equipped for cancer patients can make good use of five or more grams of radium, and no hospital which is prepared to give modern scientific treatment for cancer should have less than half a gram. Two is the rule in Belgium. Denmark, a very small country compared to the United States, has just bought five grams to add to her supply. Sweden has recently purchased six grams, thus multiplying her total by three. Australia has within the year bought 10 grams for her hospitals, and now South Africa demands that her government purchase enough radium to treat her cancer cases properly. It is a question whether American cities, states, and the General Government ought not to buy radium and loan it to hospitals, provided they will furnish satisfactory evidence that they are otherwise properly equipped to treat cancer and render periodic reports proving that they are making proper use of it. It would seem to be practicable to have this done by the Government through the United States Public Health Service or otherwise. It is probable that the total quantity of radium in the United States could be increased by 50 per cent at a cost of about two million dollars.

Up to the present, the treatment of cancer by therapeutic measures has proved to be disappointing, although now and then some surprising results have been reported. The same may be said of the serum treatment of cancer. All such methods ought to be thoroughly tried out by unprejudiced authorities and the results published. Frequently professional reticence keeps adverse criticism too long hidden, a fact to be deplored, for secrecy and quackery go hand in hand.

Aside from the prosecution of research, the strategy now being employed in the war against cancer consists in the bringing to-

gether of patients in the earliest stages of the disease and physicians who are qualified by knowledge and experience to diagnose and treat it.

There are few if any persons so expert as to be able to give the best possible attention to cancer in whatever part of the body it occurs, consequently, attention should be given to the facilities which may be afforded by hospitals for diagnosis and treatment. Clinics are being formed in many of our general hospitals. In France, Switzerland, and Belgium centers against cancer are being established, combining the most modern facilities for diagnosis and treatment, research, and the instruction of medical students. Special hospitals for the cure of cancer are not common in any country, apart from those which form the dominant feature of the European centers. There is a large one in London, one at Milan, one at Stockholm, two in New York City, one at Buffalo, two in or near Philadelphia, three in or near Boston, one at St. Louis, and one at Minneapolis. The tumor clinics brought about in general hospitals in sixteen cities of Massachusetts are a feature of great interest. They use the personnel and equipment already at hand, focusing it upon diagnosis and advice. Under some circumstances they treat cancer. Judging by the interest which these clinics have aroused throughout the country, it is probable that the example so well set by Massachusetts will be followed by other states.

There is one aspect of the treatment of the sick for which no adequate provision has yet been made. I refer to the care of advanced cases. Some general hospitals are able to keep chronic patients indefinitely, but their number is relatively small. The beds are too urgently needed for patients who can be made well. The care of incurable cancer patients is really a part of the question of the care of all chronic illness, and there are not a few who feel that special hospital provision should not be made for advanced cases of malignant disease. At the present time there are in the world about a score of hospitals for incurable cancer, all of which are supported by private philanthropy.

In the movement to improve the country's facilities for treating cancer, the existing resources, both private and public, must be materially increased. How this should be done in any given case will depend partly upon the local circumstances. As a step toward an understanding of the matter, the surveys or fact-finding investigations which have been made by some of our municipalities will be found of value. Such studies might well have a larger scope than that of a municipality. Our states could appropriately make cancer surveys. It has been proposed that the Federal Government itself undertake to find out, through comprehensive investigation, to what extent and in what ways the vast resources at its disposal can be made to help the efforts of the states and cities. There is ample precedent for the United States Government to aid in the war against cancer: it has done much to combat other diseases. Some foreign governments are doing far more than anything as yet proposed for ours. Let us not lag behind.

What, may be asked, are the organized medical profession, the public health authorities, and the general public of our country doing about the cancer problem? My answer is, They are doing a good deal, but not enough. Beginning in 1909, some of the state medical societies have had cancer committees, which have brought about the reading of papers, the writing of articles for the press, and the making of reports on various topics of importance to their members and the public. National and regional societies, including the specialists in medicine, have done likewise.

As in most civilized countries, there has been a voluntary agency in the United States

to combat cancer. For fifteen years the American Society for the Control of Cancer has promoted "cancer weeks," circulated pamphlets, prepared newspaper articles, and in other ways taught the public what have been called the "danger signals of cancer" and urged people to apply to physicians upon suspecting cancer to be present.

Recently the official public health administrators of the country have for the first time devoted their attention to cancer, and through their national organization, the American Public Health Association, have undertaken to induce state and city health departments to collect detailed cancer statistics, make microscopic examinations of tissues for diagnostic purposes, instruct the public in what they should know about cancer, co-operate in the establishment of clinics and nursing services, and in other ways give practical help in meeting the urgent needs presented by cancer in the community.

The public has not been indifferent to the cancer menace. It was the Legislature of Massachusetts, acting in response to a popular demand, that required first a survey, then the establishment of clinics and a cancer hospital. The first municipal cancer institute in the United States is the work of the Department of Welfare of New York. Private philanthropy has generously endowed research institutes, cancer hospitals, cancer wards in general hospitals, and the American Society for the Control of Cancer. There is no doubt but that a great deal more money and personal service can be had for this cause, if the ways in which it can profitably be spent are pointed out.

The last five years have seen an awakening on the subject of cancer, but the world scarcely realizes as yet the formidable character of the problem which it has to solve. In the eloquent words of Dr. Louis I. Dublin, the eminent statistician, who is, beyond most persons, in a position to speak with authority on this question, "In fighting cancer we are playing with toys when we should be using engines of a thousand horsepower."

CASE REPORT

A CASE OF RADIO-DERMATITIS

By U. V. PORTMANN, M.D., Cleveland Clinic, Cleveland, Ohio

A patient who had been operated on in May, 1928, for carcinoma of the breast with axillary and supraclavicular metastasis, had been given two courses of post-operative prophylactic roentgen therapy, with an interval of four months between the courses. On her return in January, 1929, several small local recurrent growths were present and the supraclavicular nodes were still enlarged. A slight tanning of the skin was still visible, but it was deemed wise to give another treatment in spite of the fact that a sharp reaction might be expected. In each course of treatment the following technic was employed: To the chest areas, K.V. 140, filter 4 mm. aluminum; to the supraclavicular and axillary areas, K.V. 195, filter 0.53 mm. copper plus 1 mm. aluminum. The skin received on each field 600 *r*-units.

The patient experienced a moderate systemic reaction, and two weeks later a sharp skin reaction, for which her attending physician prescribed unguentine butesin picrate. Quickly the skin reaction became very angry. I saw the patient at this time and continued the use of the ointment, since it is said to be useful for burns. The local condition became progressively worse for two days—there was great pain and burning, the redness increased, and the tissues were markedly edematous, even beyond the fields which had been irradiated. I feared that the entire irradiated area might be destroyed. I was certain that such a severe reaction should not have resulted from the irradiation alone, therefore the ointment was discontinued and, at the advice of a consultant dermatologist, we substituted Lassar's paste (original) with ichthyol (5 per cent). There was a disseminated dermatitis venenata which involved the entire body but was particularly severe within and just bordering the irradiated field. Calamine lotion with phenol (1 per cent) and menthol (0.5 per cent) was employed for this, and gave considerable relief.

At the time when the dermatitis appeared, small ulcerations were also present on the buccal mucosa. They were very painful and were thought to be some form of "angina," but they may also have been a reaction to the ointment similar to that manifested by the skin. The stomatitis was treated successfully with methylene blue (2 per cent).

After the use of the Lassar's paste the local condition began to improve immediately and within 48 hours the edema had subsided, the epidermis had begun to regenerate, and the patient was comfortable and recovering. Of course, it is impossible to state what will be the ultimate outcome, but at this time (May, 1929) the skin is apparently healthy except for a moderate tanning. It is interesting to note that the local recurrences disappeared completely and that the supraclavicular nodes are scarcely palpable.

This is a case in which radiation dermatitis was intensified by the use of unguentine butesin picrate. Though the patient may have had an idiosyncrasy for it, I find from dermatologists that unguentine butesin picrate is not infrequently irritative. I hasten to report this case as a warning against the use of this ointment for radiation dermatitis lest some one else meet with similar difficulties.

GENERALIZED OSTEITIS FIBROSA*

By I. SETH HIRSCH, M.D., NEW YORK CITY

(Concluded)

In the following case of generalized osteitis fibrosa of the cyst-forming metaplastic malacia type, the patient was studied over a period of fifteen years. He was first observed in August, 1912, at the age of 15 years.

H. W., male, born in United States, of Russian parents. His mother died of rheumatic carditis. One sister is 28 years old and has psoriasis. The patient has had three attacks of diphtheria, one of which was followed by a diphtheritic paralysis of the pharynx, from which there was complete recovery. He attended public school and was always able to keep up with his classmates in his studies. He was bright and wide awake, somewhat undersized and undeveloped and poorly nourished. There was no complaint whatsoever relating to his general health.

At the age of 8 he first noticed a slight protuberance over the right parietal bone and another soon developed in the left cheek. Both of these protrusions increased in size very slowly, and, with the exception of the deformity to which they gave rise, caused no complaint. His head had also been increasing in size since the age of 8. He recollects that at the age of 12 he had difficulty in fitting himself with clothes because of curvature of the spine. From 18 to 27 he had various fractures of ribs and clavicles. In 1920 he fractured the right femur.

The general health is good. His only complaint is pain in the sternal region and in the legs. There is defective hearing on the left side and defective vision of the right eye. The head is large—the frontal area is flattened. The left malar region is deformed and the deformation involves the whole of the superior maxillary area. There is marked rotary lateral scoliosis. The fracture of the right femur, which has united firmly, has produced a shortening of the right leg. There is deformity of the chest, due to fractures of the sternum, ribs, and clavicles.

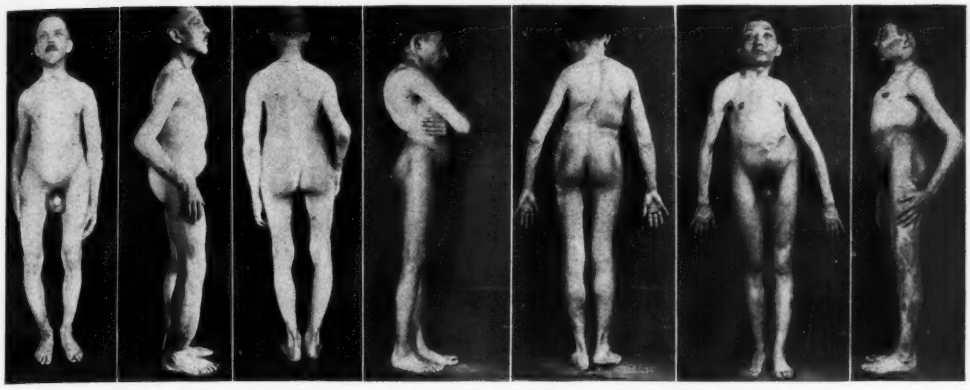
The Wassermann reaction has been repeatedly negative. The blood analysis shows uric acid 4.0, urea nitrogen 12.8, chlorides 0.49, calcium 10.9, organic phosphates 4.2. Serum calcium and magnesium values were determined before and after parathyroid injections without any change in the normal values (calcium 9.6–9.8, magnesium 1.9–1.9).

The facial and skull deformities were already apparent at the age of 8, and the spinal deformity at 12. It is justifiable to infer on the basis of subsequent studies of the slow progress of the lesion over a period of fifteen years that the changes of the disease undoubtedly existed in infancy (Fig. 17).

The roentgen examination shows the condition illustrated in Figures 18-28. These examinations have been made at three periods during fifteen years. In a general way, therefore, it may be stated that the lesions in the metacarpals and phalanges have shown a tendency to repair in the last fifteen years and that no new lesions have appeared. In other words, it would appear that the activity of the lesions in the hands had already ceased at the age of sixteen. This is borne out by a comparative study of the other bones. There is one exception, however—the skull; here the changes have been progressive.

At the first examination sixteen years ago the skull showed the following results: All the cranial bones were markedly thick-

*The first part of this paper was published in the June, 1929, issue of RADIOLOGY, pp. 505-516.



Figs. 14, 15, and 16. Generalized osteitis fibrosa showing similar head conformation and general body attitude. This is practically the same with both the Paget and von Recklinghausen type of disease. Note also the marked changes in the temporal arteries in the von Recklinghausen's disease (Fig. 32). The increase in the broad diameter of the skull and the prominent, very convex, overhanging frontal area, with the recession of the atrophic facial portion, are strikingly shown in Fig. 30.

Fig. 17. Generalized cyst-forming metaplastic malacia. Note the enlargement of the skull and the left superior maxillary area, the spinal curvature, the chest deformity, and the shortening of the right leg, due to a fracture of the femur through a cystic area.

ened, especially the frontal and occipital and superior maxillary bones. The differentiation between the tables and the diploë was lost over the frontal area, but could be made out in the rest of the bones. The occipital bone, however, showed numerous cystic areas running parallel with the curvature of the bone. The left superior maxilla and the antral region of the left side was occupied by a bony mass consisting of irregularly formed lamellæ of increased density, with widened intralamellar spaces. The entire base of the skull showed these

Figs. 18 and 19. The skull changes in the metaplastic form, similar to those of the hyperostotic type (Paget). The superior maxilla shows changes similar to those in the skull bones.

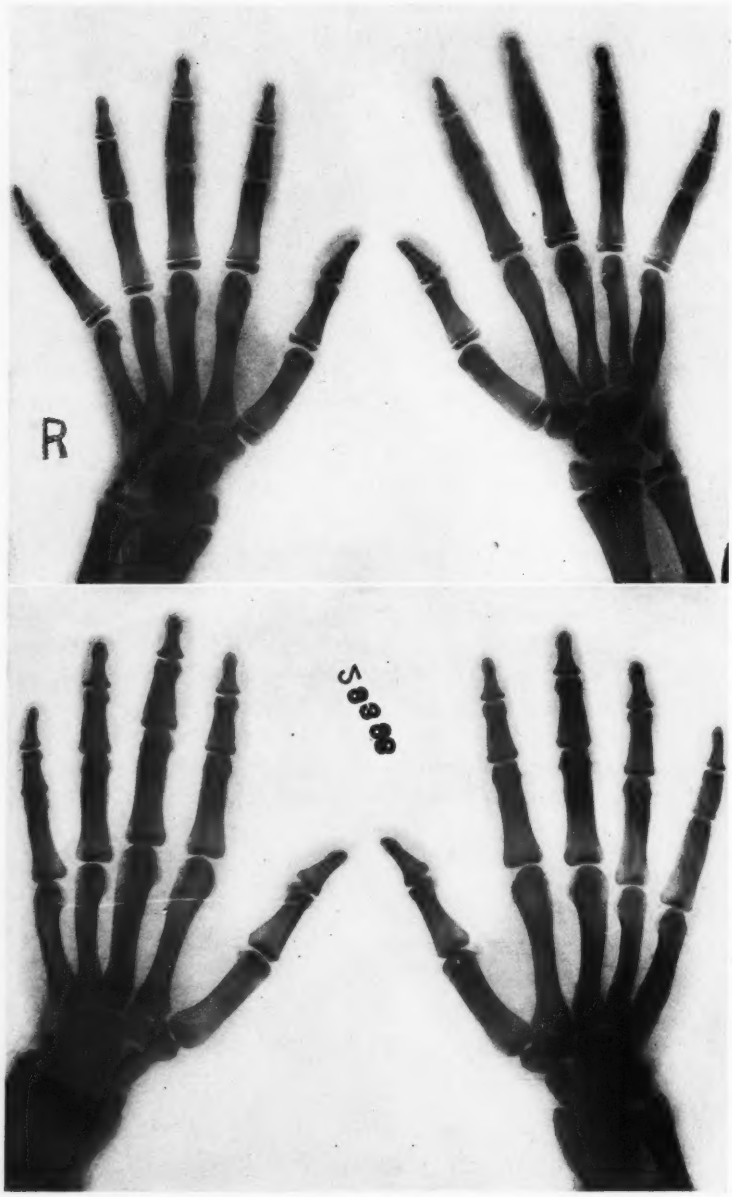


Fig. 20. Hands of 16-year patient. The bones of the hands illustrate every variety of lesion found in generalized osteitis fibrosa. *Right hand:* Middle metacarpal shows diffuse endosteal new bone production proximally but thinning of the cortex and expansion at its distal end, due to widening of the medullary areas. This is a change characteristic of a late stage of this disease. The second, fourth, and fifth metacarpals show a lamellation of the cortex and the changes in the cancella, due to breaking down of the interlamellar septa. This is the early stage of the disease. These changes are most marked in the proximal end of the diaphyses of the bone. The head of the first metacarpal shows changes in the distal cancellated area. The proximal and middle phalanges of the middle finger show small "cystic formations" with well

(Conclusion of legend of Figures 20 and 21, on opposite page.)

formed trabeculæ, the base of the middle phalanx being expanded. *Left hand:* The fifth metacarpal shows a large single cystic area, well circumscribed at the base, with definite expansion of the shaft. The cortex is expanded, thinned, and striated. There is endosteal bone formation in the base of the fourth and in the head of the first metacarpal. Medullary and cancellous changes are shown in the base of the second and third metacarpals. The cortex of nearly all the metacarpal bones shows striation and lamellation. There is a small cystic area in the distal end of the proximal phalanx of the fifth finger; the middle phalanges show expansion, striation of the cortex, and changes in the cancellous tissue. There is "cystic" degeneration in the middle phalanx of the second finger. The terminal phalanges are pointed and the terminal tufts are not enlarged. A noticeable feature is the flaring of the metacarpals and the phalanges at their juxta-epiphyseal ends. The epiphyses are apparently well formed; the epiphyseal line is regular. There is a defect in the epiphysis at the base of the middle phalanx of the right hand (*above*).

Fig. 21. An examination of the same patient seven years later gave the following results: The changes visible in the previous examination can still be definitely recognized. The areas of the cortical thickening in the middle metacarpal of the right hand show increased density and the medullary area can scarcely be made out. Some of the small "cystic" areas remain, others have been filled in by new bone formation (fourth right metacarpal, third right proximal phalanx). (*Below.*)

productive changes. The sella turcica was very small and its walls were thickened. There was an upward displacement of the posterior portion of the base of the skull, particularly in the region of the foramen magnum.

With increasing age the skull changes have progressed so that now every part of the cranium is involved. The differentiation between the outer table and the diploë is lost. The inner table is, however, clearly defined. Irregular masses of bone are scattered throughout the widely expanded cranial bones, and the bones are porous, due to numerous irregular areas of osteolysis, which, however, seem to be far more numerous on the outer surface of the thickened bone.

Pelvic examination showed cystic areas in both iliac bones, in the wings, and in the sacral portions. The ischium and pubis on the right side showed a loss of cancellated tissue and large cystic areas without bone expansion.

There has been considerable increase in the extent of these areas in the last fifteen years. They are still well defined, without evidence of healing, with well marked trabeculæ, and the thickened, striated cortex of the ilium has become thinned, as a result of the enlargement of the cystic areas. The ischium and pubis show diffuse cystic changes.

Marked progressive changes had taken

Fig. 22. Metaplastic changes in humerus; cystic changes in ribs and scapula; marked malacic changes in the vertebræ.

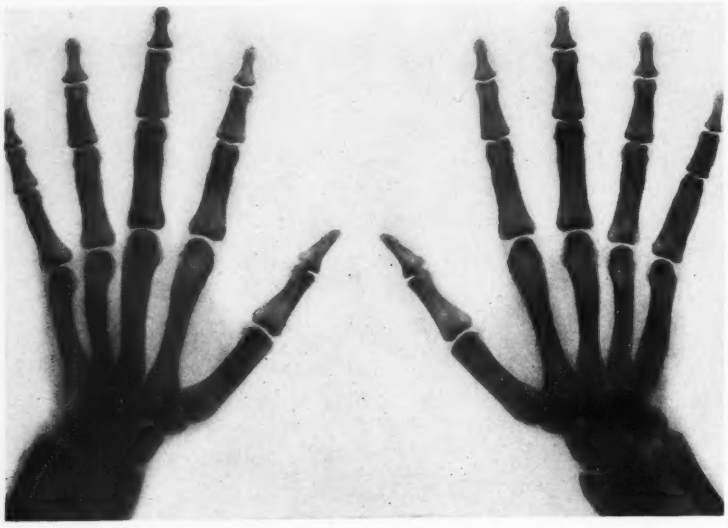


Fig. 23. Same hands (as shown in Figures 20 and 21) at 31 years show a still further progress of the lesion in the direction of what might be termed repair or healing. Most of the so-called "cystic" areas have filled in with dense bone. The sclerotic changes in the middle metacarpal of the right hand have not altered. The cystic area of the bones of the second metacarpal of the left hand is still clearly visible. The small cystic area at the distal end of the proximal phalanx of the fifth finger of the left hand has lost its definiteness. The cystic area at the base of the fifth metacarpal on the left hand shows complete ossification. The metatarsals and phalanges show similar changes with a similar genesis.

place in the femora sixteen years ago. There was on the right side a definite coxa vara, with thickening of the neck, which, with the trochanter, contained small cystic areas. At the last examination the medullary cavity is very wide, the cortex being very thin. There is a large cyst in the neck. The coxa vara is well marked. There is an old oblique fracture at a point two inches below the lesser trochanter, extending obliquely downward and outward and with inward displacement of the lower fragment and union with deformity. The fracture had evidently taken place through a huge cystic area. There have been no marked changes on the opposite side. The upper end of the left femur, including the neck, shows numerous cystic areas, with considerable trabeculation and ridging. The lower end shows irregular calcifications in the medulla.

There is marked rotary lateral curvature of the spine, with hyperostotic and cystic changes in the vertebræ. The bodies of the lower vertebræ are flattened and widened.

The ribs show considerable expansion and cystic areas, with well formed trabeculæ.

The entire lower two-thirds of the scapula is converted into a series of well defined bony cavities, with thin trabeculæ.

The changes in the long bones, tibiæ and fibulæ, show no striking difference in their general conformation in the fifteen years. The lesions present when the case was first examined can still be made out in the most recent examinations without any marked variation, without such variation as is present as the result of effort at re-ossification and the tendency toward healing.

This case is by no means unique. There are other cases in the literature in which at this age or much younger a localized le-

Fig. 24. Left femur showing fracture through cystic area. Cystic changes in upper fragment and coxa vara, due to malacia of the femoral neck.

Fig. 25. Right femur showing widened medulla and numerous cystic changes in trochanter and shaft.

sion, a protuberance in the facial or skull bones, or a deformity or fracture of the long bones, has directed attention to the disease, which gradually progressed to complete and general bone involvement.

In a case reported by Stack (499), the remarkable specimens of which are in the Bristol Museum, the skull attained a thickness of three inches; the patient was a female, who died at the age of twenty-one. The enlargement of the head was first noted at seven years. There had been a fracture of the femur. The changes of osteitis fibrosa were not limited to the skull, but the femora, pelvis, and right tibia were involved, though vertebræ and ribs were normal. It is inconceivable that even under such circumstances the head deformity could have sprung into existence suddenly. It is logical to believe that it was several years before the bone changes attained sufficient prominence to attract attention.

The period of clinical latency, the periods of apparent inactivity, and the gradual development of the changes when the disease is progressive, would tend to bring the time of onset back to the earliest periods of childhood.

There is another factor which tends to support this. If the photographs of an individual with this disease are studied, certain stigmata of the disease may often be

Fig. 26. Cystic formations in the pelvic bones. The iliac bones contain the large cysts. The ischium and pubis also show cystic changes.

noticed, in the size and shape of the head, conformation of features, low position of the ears, or in the peculiar bowing of the

Fig. 27. Lateral view, showing the fibro-cystic changes in the fibula.

Fig. 28. Simple unilocular solitary metaplastic malacic cyst in upper end of fibula, oval, circular, or irregular in shape, with thickened marginal zones and a bulging cortex. The interior of the area is usually homogeneous and transparent, frequently with single dense bands due to remaining bone trabeculæ or septa. It may be multilocular, having a sponge-like appearance, with numerous rounded or oval areas of varying size separated from each other by thin septa. The sites of predilection are the shaft end of such bones as the proximal humerus, the great femoral trochanter, the fibula, and tibia. Extension into the epiphyseal cartilage does not occur as a rule.

legs, as far back as such photographs are obtainable, even into childhood.

The early origin of generalized osteitis fibrosa may be inferred from another consideration. Paget said in 1889, after he had seen 23 cases: "I have tried in vain to trace any inherited tendencies to the disease; I have not found it in two members of the same family."

But since that time instances of more than one case in a family have been reported, suggesting that heredity exerts a striking and important influence in development of generalized osteitis fibrosa. These cases are all reported as instances of Paget's disease, and from the description the inference is jus-

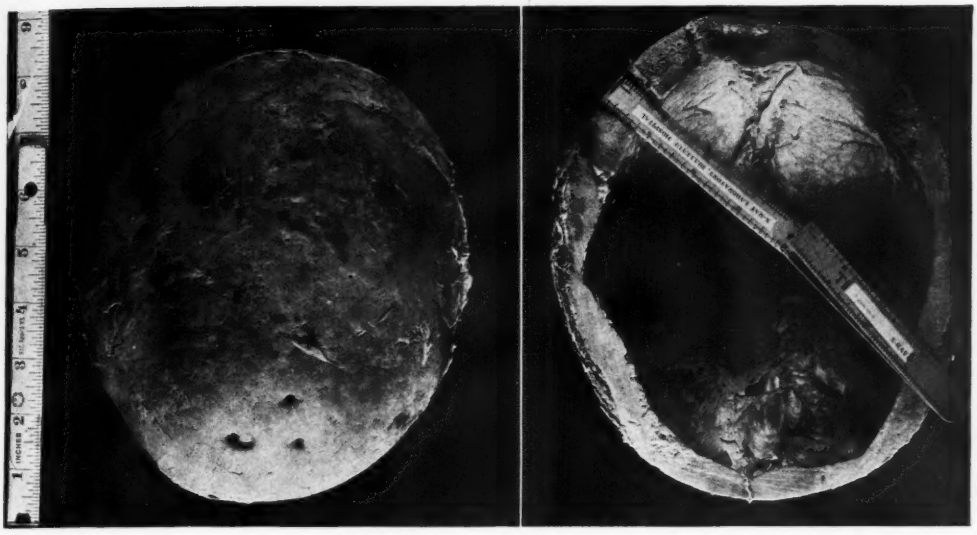


Fig. 29. Inner surface vault of skull showing the thickening of the skull bones, the deepened vascular grooves, the marked irregularity of the inner surface and the numerous foramina in the outer surface.

tifiable that these were really of this variety of generalized osteitis fibrosa: Manwaring-White (341), two brothers (radiograph); Oettinger and Agasse-Lafont (384), father and two sons out of three children; Lunn (330), two brothers, 68 and 65 years; Higbee and Ellis (225), mother and son; Smith (494), father, 74, and son, 42; Chauffard (90), mother and daughter; Berger (48), mother and son; Kilner (259), brother and sister, 69 and 60 years; Robinson, two members of a family; Pick (407), father and daughter; Hurwitz (231), mother and son; Parry (392), two sisters (three brothers normal); Abbe (1), two brothers; Da Costa (106), two sisters; Forssell (157), two brothers about 60 and sister 55.

The cases reported are by no means an indication of the actual rôle which heredity plays in this disease, for many of the family histories are not given or are incomplete in the numerous cases. The story of the patient is of no value. It is essential to examine all the members of a family, for, to the patient, deformities of the disease may pass as a familial resemblance. The determination of this point must be based on roentgen examination—the only positive test. Absence of the disease in other members of the family cannot be accepted according to modern criteria unless a roentgen-ray examination has been made. I believe that when the families of patients with Paget's type of generalized osteitis fibrosa are submitted to roentgen examination, this hereditary tendency will be established by a number of instances. It is important to remember that the transmission of this disease is subject to all the laws, exceptions, and modifications of hereditary diseases. Such a theory would also explain the presence of the typical changes in but a single bone.

How does this type of generalized osteitis fibrosa manifest itself in early life? No cases with the typical adult changes of hyperostotic metaplastic malacia (Paget) have been found in infancy or childhood. What is the appearance of the incipient changes in this disease? The answer must be sought in the hyperplastic types of malacia.

Fig. 30. See legend under Figures 14, 15, and 16.

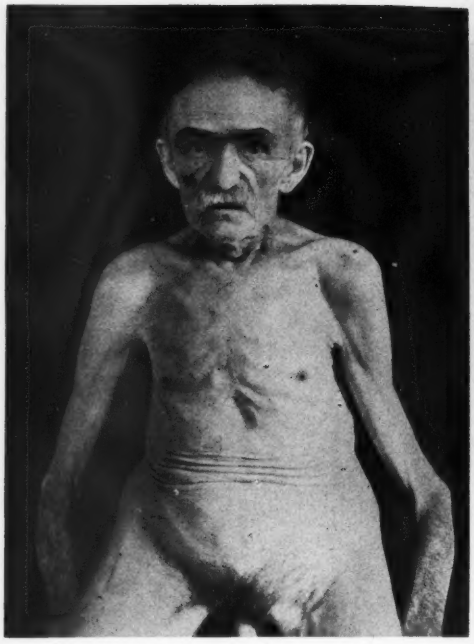


Fig. 31. See legend under Figures 14, 15, and 16.

Cases of this type are the forerunners of what later in life is easily recognized as Paget's disease. However, in its incipient stage the bone changes of this type of disease differ from that of the adult type of lesion, and are not those familiarly known. When the disease is well advanced and has existed for many years, it is easily recognized; but the changes in their earliest phases, before marked demolition of the normal bony structure has taken place and before marked restorative changes and deformity have appeared, are not so well known and are of the type which may, in von Recklinghausen's classification, be placed under hyperplastic malacia.

It must be remembered that the bone changes of hyperostotic metaplastic malacia, as usually seen in the adult, are far from uniform. Most commonly the cortex and cancellous bone are both porous and sclerosed. This is the late stage of the disease, when the whole architecture of the bone is altered and the distinction between compacta and cancellous bone is lost and the medulla is occupied by calcified fibro-osteoid tissue.

The softening, due to bone resorption and fibro-osteoid tissue substitution, however, is a prominent phase when the disease enters its adult stage. The softening leads to bending. The amount of deformity is dependent on the extent and severity of this phase. If the patient should die at this stage, the microscopic changes would show

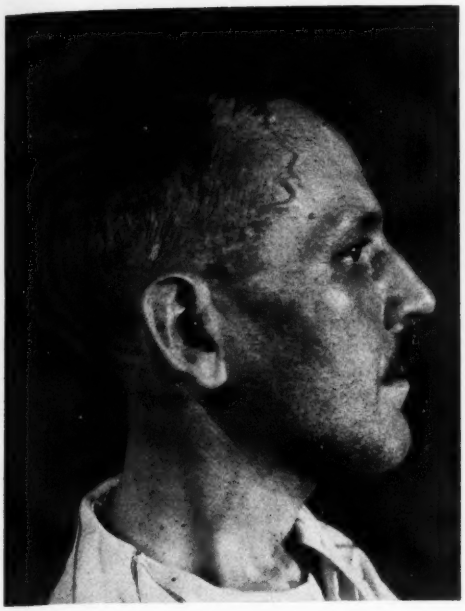


Fig. 32. See legend under Figures 14, 15, and 16.

Fig. 33. Metaplastic malacia, with marked bowing of the humeral neck as a result of malacia and deformity resulting from an old healed fracture. The lower portion is undergoing cystic degeneration.

bone resorption and fibro-osteoid tissue formation, with little calcification of the osteoid tissue. The process is a slow one, unaccompanied, as a rule, in this early stage, by even minimal fractures of new calcified trabeculæ. Later on, however, the new osteoid tissue becomes calcified and the bones become fixed and hardened in their deformed shape and thickening is produced by new bone laid down on both old and new trabeculæ and under the periosteum, producing the classical picture of the adult lesion.

That the cystic metaplastic type makes itself grossly manifest earlier is due to the lack of bone regeneration and the predominating fibroid and fibro-osteoid changes. Thus there is early bowing and bending due to the malacia and cyst formation due to the degenerative changes.

On the other hand, the regenerative phase, the new bone formation, may go hand in hand with fibroid metaplasia, and, for a considerable period of time, the integrity of the bone structure be maintained. However, this compensatory effort finally fails to maintain the balance, and then the deformity becomes apparent. By this time,

Fig. 34. Hyperostotic form of metaplastic malacia of the humerus. There is an old fracture through the neck.

Fig. 35. Various lesions found in the bones of the hands in generalized osteitis fibrosa.

however, the individual has attained maturity and thus the disease is thought to have had its incipiency in adult life.

Generalized hyperplastic malacia in three children (two boys and a girl) in the same family.—The children are now nine, eleven, and sixteen, respectively. When I first saw them three years ago they showed practically the same lesions as are now present. In three years the lesions have not changed to any marked degree. The father, 44 years of age, was born in Italy and is a mason by occupation. The mother, also born in Italy, died of nephritis five years ago at the age of 42 years. The parents came to this country fifteen years ago. They had nine children, eight of whom are living, and three of them have the disease.

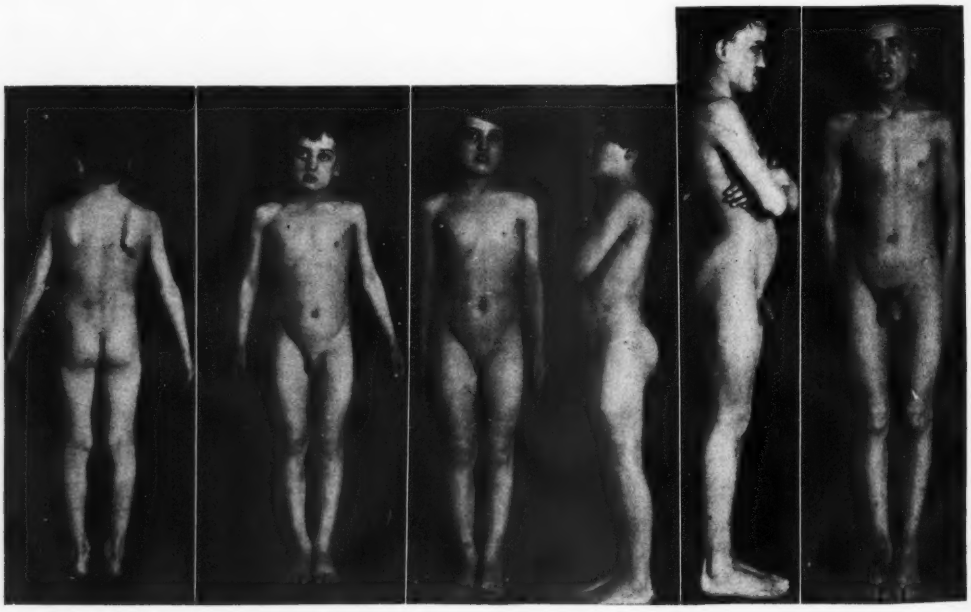
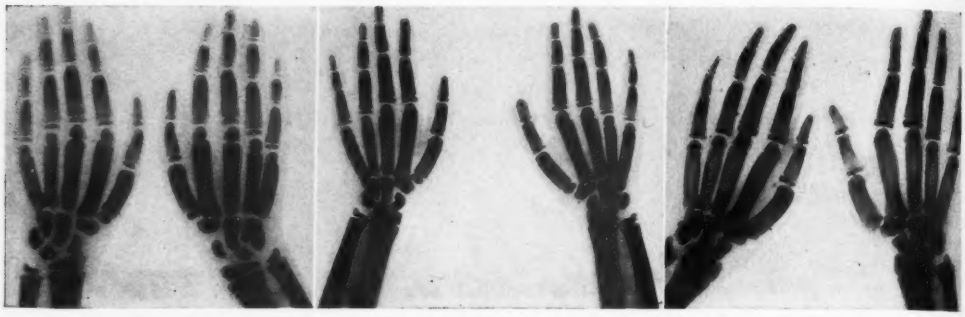


Fig. 36. Generalized hyperplastic malacia. Three children in one family.

Tony	Carmella	Ralph
Age nine years and nine months.	Age eleven years and seven months.	Age sixteen.
Mother's pregnancy and delivery were normal.	Mother's pregnancy and delivery were normal.	Mother's pregnancy and delivery were normal.
Normal.	Normal.	He was a full-term baby. He began to walk at one year and to talk at two years.
Began having spasms when one year old. He continued to have them; has had at least one a year; the last one, about six weeks before this report. These spasms start and end within a week. The face remains out of shape for this length of time. The spasm may come during the night from no known cause. There has never been any unconsciousness. There are no indications of the difficulty other than facial expression.	She started to have spasms at the age of ten or twelve months. She had them about every six months or every year until about two years ago. These were characterized by twitchings and spasms without unconsciousness.	He was nearly a year old when he started to have spasms. He continued to have them about once a year, until the age of eleven years.
No other illness.	She has had no other illnesses.	He had pneumonia when he was two years old. He has had no other illnesses.
Physical examination shows: square head; heavy-set features; teeth irregularly developed; speech defective; no paralysis or atrophies; .cranial nerves intact; heart, lungs, and abdominal viscera negative; superficial and deep reflexes intact; gait good; well developed; well nourished; good musculature.	Physical examination shows: coarse, heavy-set features; webbed fingers—marked on left hand; fingers of left hand bent; notched teeth; speech defective; cranial nerves intact; no paralysis; heart, lungs, and abdominal viscera negative; superficial and deep reflexes intact; gait good.	Physical examination shows: defective speech; slight webbing of the fingers; contraction of fingers; face is broad and coarse, with heavy-set features; eyes wide apart, but do not slant; mouth is kept open; the bridge of the nose is not depressed; beads of perspiration on nose and upper lip; lower jaw large and heavy; cranial nerves intact; ears are prominent and large; lungs and abdominal viscera negative; liver and spleen not enlarged; superficial and deep reflexes intact; station and gait good.
Wassermann reaction negative. Blood examination normal.	Wassermann reaction negative. Blood examination normal.	Wassermann reaction negative. Blood examination normal.
See Fig. 36.	See Fig. 36.	See Fig. 36.

The bones in all these children show similar changes (Figs. 36 to 42). The predominating change is a hyperplastic and hyperostotic process, with thickened cortex and enlarged diaphyseal ends. The epiphyseal development is normal. The bones of the legs and arms are bowed. There is also bowing of the first matacarpal bone. The cortex is symmetrically thickened, while the medulla is everywhere clearly defined and in many bones widened and the cancellous structure is everywhere normal but shows an increased calcification. All skull and facial bones are, however, thickened so that the diploic layer cannot be seen. Though the bones of the base of the skull are thickened the sella turcica is of normal size; the clinoids are sharp. The vascular grooves are deepened. There is a tendency towards flattening and broadening of the diaphyseal ends at the epiphyses, which gives the metacarpals and phalanges a clubbed appearance. There are minute localized "cystic areas" in the cortex. Everywhere, how-



Figs. 37, 38, and 39. Hands of the three children (Fig. 36), showing relatively normal carpal development. There is a peculiar shape to the phalanges and metacarpals due to widening of the distal ends, the thickening of the cortex at the distal ends of the phalanges being marked. The terminal phalanges of the index fingers of the female child show perforations. The terminal tufts are undeveloped.

ever, the architecture of the bone is still definitely maintained and the lamellæ are still regular and orderly in arrangement.

The so-called osteopetrosis or "marble bones" is a lesion which may be classified as a hyperplastic phlegmatoplastic malacia. In osteopetrosis the diaphysis and the metaphysis have a homogeneous density without differentiation of the cortex, spongiosa, or the medullary canal. This is generally obliterated, the bone being solid and structureless. There are encircling bands of lime salts parallel to the epiphysis and isolated areas of calcification. The whole bone is changed into a compact structureless mass. Fractures are common. The changes are very marked about the sphenoidal sinus, which may be obliterated. The sella turcica is deformed, diminished in size, and the clinoids are thickened, clubbed, and enlarged. There are also blood changes.

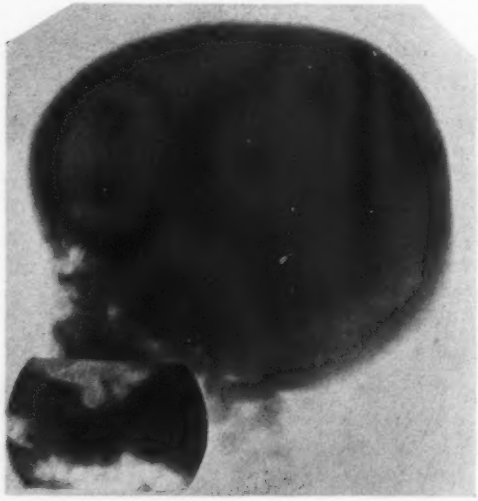
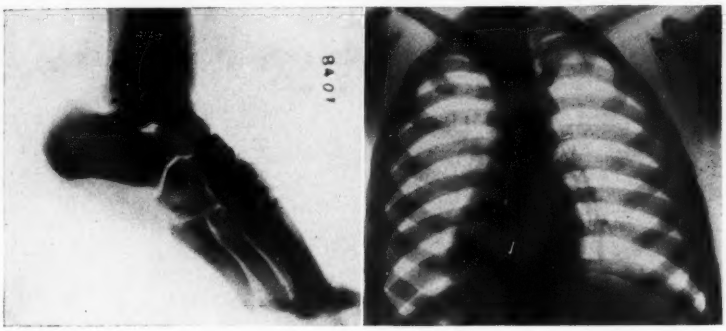


Fig. 40. Skull of female child, showing hyperplastic and hyperostotic changes in both the vault and base. The inset shows the thickening at the base, the clear-cut sella turcica (uninvolved) and the sharp posterior clinoids.

The generalized obliteration of the bone marrow in severe examples of this disease results in a loss of its hematopoietic power, with a resultant metaplasia of hematoplastic elements into other organs, the spleen and liver resuming their fetal blood-forming function. Thus there are found associated with the bone changes, enlargements of the spleen, liver, and lymph glands, with metaplasia of blood-forming elements, more often myelocytic. The blood picture and autopsy findings may be the general picture of the severe anemias and the leukemias. No such changes are present in the above cases.

It is the less marked cases, those with partial involvement, which give signs that permit the grouping of this disease with the malacias. There are isolated areas of calcification, alternating with osteoporotic bone. It is at these points where the bowing is marked. Certain bones, like the clavicle, may remain uninvolved.

The changes may involve only part of the bone, a definite line of demarcation being present between the normal and involved bone. Such demarcations are not unusual in osteitis fibrosa. On one side of this line, all traces of the medullary canal may be lost. On the other side, the medulla is clearly shown; the cancellous structure is normal but somewhat more dense than usual. Such findings would indicate a primary malacia, with porosis, and a secondary hyperostotic change, which in its essential features is the genesis of all types of generalized osteitis fibrosa or the hyperostotic metaplastic malacias.



Figs. 41 and 42. Ankle and chest, showing the well-marked cancellated structure everywhere definite but of increased density. The cortex of the metatarsals is wide and dense, but the medullary cavity is not encroached upon.

PART III

The connection between the hyperplastic and metaplastic types of malacia is shown by the mixed forms. Here both types of lesion are found.

METAPLASTIC AND HYPERPLASTIC MALACIA

The patient whose case is reported is a male, white, unmarried, age 32 years. Mother died during his infancy. No brothers or sisters. Can tell nothing about father. Childhood, sickly. When 10 years old, had trouble with left hip. After a period of several weeks in extension, he left the hospital with a brace and high shoe, which he wore for several months. When 14 years of age, he had an attack of severe pain in the left leg and hip, and was in St. Mark's Hospital, where the peculiar shape of his legs and the lumps in buttocks and thigh were first noticed. At 19, following a perforating wound, he developed an infection of the foot and leg which lasted for four weeks. This was followed by contractures of knee and foot. He entered Bellevue Hospital very soon after this for treatment of the contractures. He was put up in extension for five or six weeks but was compelled to return again two weeks after discharge because of severe pain in knees and hips. This time he remained for five months, when he was discharged as "cured." When he entered the hospital a third time, a specimen was removed from the masses in his thigh for diagnosis. The report returned from the Loomis Laboratory was "sarcoma." He was advised to have the masses removed for the purpose of making a toxin, which he refused. The lumps in the buttocks and thigh had never given him any pain: they had increased in size. A month later, a section from the mass in the right thigh was removed, and, upon exam-

Fig. 43. Combined hyperplastic and metaplastic malacia. Side view of the lower extremity in slight flexion. The protrusions are due to the osteochondromata in the buttocks and trochanter as shown in Figure 45-A. The osteochondromata are a complicating factor in this disease, the results of cartilaginous dysplasia.

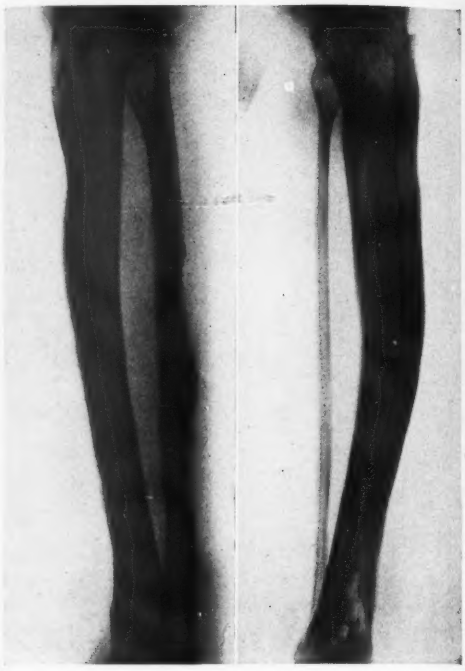


Fig. 44. Combined hyperplastic and metaplastic malacia. The metaplastic changes and the resulting malacia are shown in the bowing of the bones of both legs. The hyperplastic changes are shown as longitudinal bands of new dense bone, producing irregular thickening of the cortex (melorleostosis). At the lower end of the right tibia cystic changes are apparent. The hyperplastic process when marked is sufficient to distort the contour of the left fibula and to produce periosteal osteophytes (see scheme, Table II). The right fibula is involved in its lower third, but only slightly. There were similar metaplastic changes with cyst formation in the tarsal and metatarsal bones.

ination, it was reported "sarcomatous." He then underwent treatment with Coley with toxins for ten weeks, at the end of which time he refused further injections. Six months later an ulcer broke out on the inner side of the left ankle. Mercurials and iodides were used extensively for about ten months without much apparent improvement or relief. During convalescence and recovery from German measles the ulcer healed. A year later another ulcer appeared, an inch above the old one. This one took about one and one-half years to heal. At the age of 25, he went to the Presbyterian Hospital, and during his stay there he was given several doses of salvarsan. Another piece of tissue was removed from the mass in his thigh and the pathological report returned stated that the mass was mostly car-

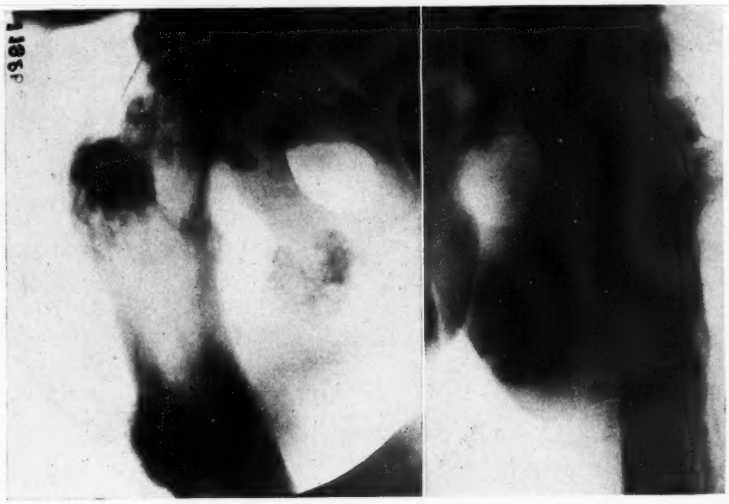


Fig. 45-A. The hyperplastic changes are shown in the marked thickening of the cortex, irregularly distributed as longitudinal bands along the femora. There are large osteochondromata springing from the junction of the ischium and pubis, from the greater trochanters, and from the upper third of the left femur.

tilage. Later, in 1911, the hamstrings at the right knee were cut for marked contractures. Since the operation the knee has been ankylosed. Had Neisserian infection five years ago. Has had eight Wassermanns taken, all of which have been negative. A Wassermann was taken with a highly sensitized antigen, but reacted "slowly negatively." Two years ago he underwent a course of mixed treatment in Bellevue, without benefit (Fig. 43).

Physical Examination.—There is marked limitation of motion in all joints of the lower extremities, with both knees in slight flexion and adduction. There are movable masses in both buttocks. The radiographic examination from the pelvis down shows changes in the bony structure of all the bones of the lower extremities. From the pelvis up the bones are all normal. There are osteochondromatous masses springing from both acetabula, the left trochanter, both ischio-pubic junctions, and from the upper part of the left femur. These are congenital in origin and have no direct association with the generalized bone lesion, though cartilaginous dysplasia is not a rare accompaniment of the various forms of generalized malacia. The hyperplastic process is shown

Fig. 45-*B*. Combined hyperplastic and metaplastic malacia. Knee joint showing widened medulla of lower end of femur, thickened cortex, cystic changes in patella and upper end of tibia. There is obliteration of the knee joint space.

Fig. 46. Cyst-forming metaplastic malacia. This lesion appears to be entirely confined to the left lower extremity. There was much shortening of the entire lower extremity. The demarcation between the involved and normal areas is sharp and bounded below by the epiphyseal line. Note the changes in the upper end of the tibia and the lower end of the tibia and fibula. The cortex of the bone in the upper end is very thin and there are several oval cyst formations, longitudinally disposed, separated by thin septa. In the lower end of tibia and fibula the defects in the bone shadow merge with the epiphyseal line. The defects are sharply limited on the diaphyseal side. The appearance suggests the presence of cartilaginous foci and is an earlier stage of the lesion in the upper end of tibia, where the ossification has completely enclosed these islands, which have been transformed into cysts. There were also changes in the lower and upper ends of the femur and in the head of the first metatarsal bone. The involved bones of a case reported by Burchard are shown at (*A*). Note the similarity of the lesions. The right tibia and fibula (*B*) show no abnormality aside from healed rickets. Note the flaring diaphyseal ends.

by an excess of new subperiosteal bone production in irregular longitudinal bands[8] and the metaplastic process by the bone expansion, the cyst formations, and by the malacia (Figs. 43 to 45-*B*).

[8]This lesion has been described under the names of "mélorhéostosis," "osteosclerosis," "osteosis eburnisans monomelica," and "osteopathia hyperostotica." It consists of a very compact hyperostosis, which occupies the whole length of the bone as an uneven sheathing, or as it has been termed, "a flowing hyperostosis." It may be limited to parts of a single bone or may involve all the bones of an extremity. These longitudinal bands of hyperostosis are irregularly disposed over any aspect of the bone. The contours are irregular and the deposit is not merely subperiosteal but cortical. In this apparent irregularity of distribution, there is, nevertheless, something which suggests a definite distribution, which does not, however, correspond to any nerve or vascular supply. The disease can be traced back in its manifestations to the period of childhood. It is relatively slow and painless in its development in the earlier periods. It is evidently associated with a congenital defect in bone development.

CYST-FORMING METAPLASTIC MALACIA

Another variety of generalized osteitis fibrosa is the disease described by Mikulicz (351) in 1904 as osteodystrophia cystica juvenilis. Bockenheimer suggests that this name should be discarded. The changes are

Fig. 47. Cyst-forming metaplastic malacia, adult type. There are numerous cyst formations at the head of the humerus. There are also cystic changes in the acromial end of the clavicle and in the acromion. There is a large cortical cyst formation below the head of the fibula and a small cyst at the lower end of the tibia. Note the numerous cyst formations in the opposite tibia: these appear to be cortical in origin. There is marked atrophy of the phalanges, with changes in the medulla, and a peculiar shortening and outward flaring of the terminal phalanx of the thumb. There is symmetrical thickening of the cortex of the second and third metacarpals and irregular cortical thickening of the neck of the fifth metacarpal. (See Figures 48, 49 and 50.)

Figs. 48 and 49. See Figure 47.

those of localized osteitis fibrosa with cyst formation.

It is, apparently, a congenital lesion of the metaphyseal ends of the long bones. The lesion is usually solitary, localized, and sharply delimited from the normal bone, but in rare cases may be multiple. The lesions have the histological characteristic of osteitis fibrosa of the metaplastic type of von Recklinghausen.

Von Mikulicz considers this disease as a disturbance in the development of growing bone, the intensity of the changes varying directly with the rapidity of growth.

Adjacent to the cyst formations or in other sites, there are undeveloped stages in which the bone tissue is displaced by fibrous tissue (osteoid) and richly cellular areas of giant cells. The disease is benign, there being no metastasis or recurrence after operation. Of the four cases reported, two had isolated cysts in the upper end of the humerus; one, an isolated cyst in the upper end of the femur; one, both femora and tibiæ, and in one case both femora, the upper end of the radius and the tibia were all cystic.

There is a congenital lesion of the juxta-epiphyseal ends of the bones, both long and flat, characterized by defects in the bone formation. These defects in the bone are,

Fig. 50. See Figure 47.

so far as can be judged by the radiograph, due to the interposition of masses of cartilage, which, as growth proceeds, are enclosed and separated from each other by bone septa. The epiphyseal cartilage develops normally as growth proceeds. The diaphyseal ends of the bone assume an appearance of strips of diminished density devoid of bone markings, separated from each other by well formed bony ridges or septa. These defects in the bone shadow are probably due to cyst formations resulting from the degeneration of the cartilaginous inclusions, or to fibro-osteoid changes (Fig. 46). Ossification—either granular or massive—does not take place, and the counterpart of the lesion may be seen in adults (Figs. 47 to 50). All the bones may be involved, including those of the skull and pelvis. On the other hand, the lesions may be confined to the bones of one extremity, most commonly one of the lower, and the pelvis.

The cystic degeneration extends to the epiphyseal line, which is not encroached upon. The epiphysis, when ossified, may show osteoporosis. The cysts are cortical in origin. The changes result in a deformation of the end of the bone due to enlargement and bowing. In the femora they are normally located in the neck of the bone, extending in the direction of the lesser trochanter and shaft. In the long bones they are longitudinally disposed. There may be shortening and bowing of the involved bones.

Ollier, in 1898, described a condition characterized by irregularity and retardation of ossification at the epiphyseal cartilage. The cartilage does not undergo the normal ossification for a long time and then most irregularly. The cartilaginous nodules may be subperiosteal or medullary. The extremity involved is, therefore, retarded in growth.

Ollier lays stress on the roentgen appearance of rarefied areas with denser areas scattered throughout. It may be seen in the phalanges and in the juxta-epiphyseal ends of long bones. The striated, rarefied areas in the diaphyseal ends of long bones, reported as the changes of Ollier's disease, are really cases of generalized osteitis fibrosa of the type above described.

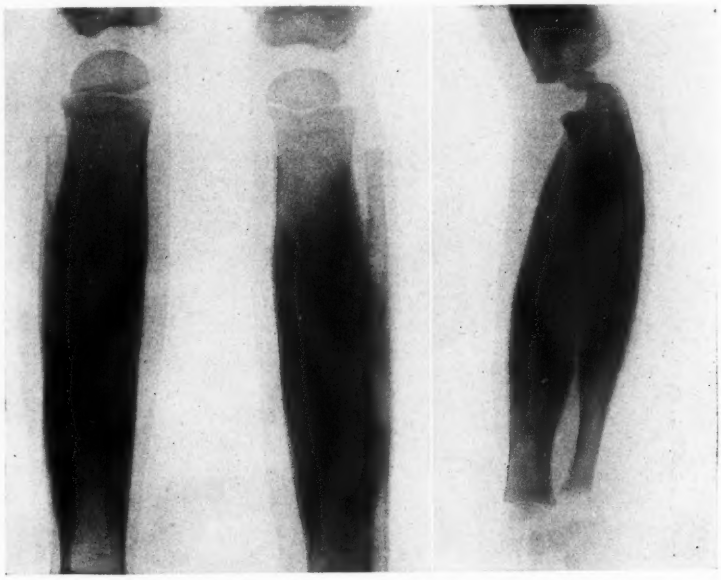
CYSTIC METAPLASTIC MALACIA WITH HYPEROSTOTIC CHANGES[9]

Infant, first examined at the age of one year, and deformities noted. Syphilitic nature of the disease was completely ruled out. The general condition of the child was good.

Roentgen examination shows the follow-

[9]This case is reported through the courtesy of Dr. Lewis Gregory Cole.

ing changes (Figs. 51 to 55): The skull is enlarged. The differentiation between the external and internal tables is obscured by the disappearance of the diploëtic layer, except in the region of the anterior fontanelle, where it is markedly widened. There is a slight depression over the anterior fontanelle. The frontal bone in its lower part is over 1 cm. thick. The inner table can be made out, the outer table cannot. There are small cystic areas in the thickened bone. The orbital plates are thickened. There is also sclerosis of the petrous and mastoid portions in the temporal bone and of the sphenoid bone around the sella, which is of normal size, and open. The skull changes appear to be similar to those seen in adult cases of generalized osteitis fibrosa. The dentition is apparently normal.



Figs. 51 and 52. (The following description of this case applies also to Figures 53, 54, and 55.) Metaplastic cyst-forming malacia (congenital). (Courtesy of Dr. Lewis G. Cole.) There are large diaphyseal cyst formations associated with hyperostotic changes. All the long bones are involved. There is thickening of both tables of the skull, but the frontal area shows the appearance characteristic of true hyperostotic malacia.

The left femur shows a spindle-shape enlargement extending from a point 2 inches below the hip joint to a point 3 inches above the knee joint. There is lamination of the cortex, widening of the medulla, and a considerable new bone production irregularly distributed, suggesting a multilocular cyst formation. Similar expansion involves the mid-portions of the diaphyses of the tibia, fibula, radius, and ulna. The contour of the cortex is irregular. The epiphyseal lines in the upper and lower ends of the tibia appear to be regular and normal. The cortex of the metacarpals is very thin and the cancellated tissues show atrophy and the formation of small cyst-like areas due to absorption of the lamellæ. The small area of sclerosis can just be seen in the first metacarpal bone.

CYST- AND TUMOR-FORMING METAPLASTIC MALACIA: METAPLASTIC NEWGROWTHS

The lesions were found in the long bones and in the skull. One of the tumor masses

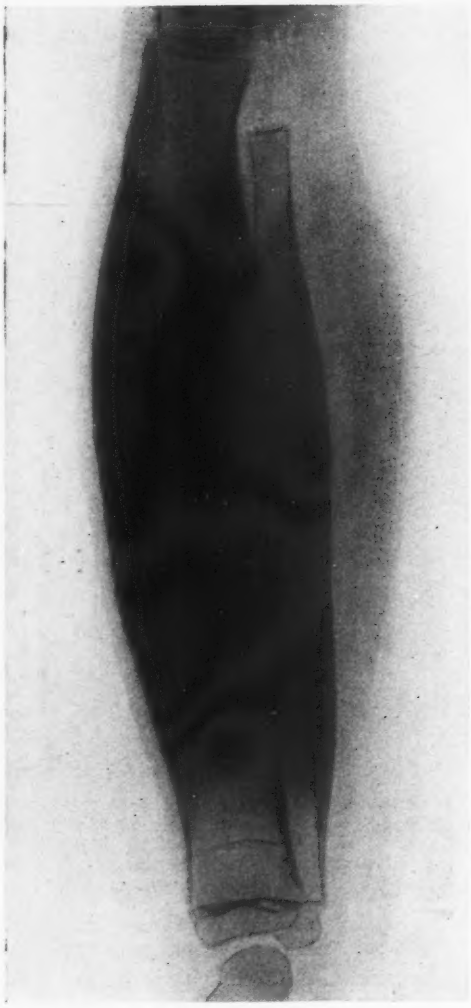


Fig. 53. See general caption which applies to case under Figures 51 and 52.

Fig. 54. See general caption which applies to case under Figures 51 and 52.

was removed and a pathologic examination showed giant-cell tumor. Other tumor masses healed spontaneously. (Figs. 56 to 66.)

This patient is a female, aged 22 years.

Family History.—Father living and well; mother died of tuberculosis seventeen years previously; two brothers living and well. Father has had diabetes. Mother had syphilis.

Personal History.—Did not have the ordinary diseases of childhood; has always been well and active. She has had a chronic discharge from the right ear since childhood. No evidence of acquired syphilis.

Present Illness.—About a year previous to examination had aching pains in arms and legs, in the left side, and in the lower dorsal and upper lumbar region. These continued to increase until she became quite invalided. A year previous to examination a lump appeared over the right tibia. Seven months previous to examination a similar one appeared over the spine, at the level of the scapula. These points were painful, but the pain was not so marked as it had been in the beginning.

Chief Complaint.—Pain in the upper portion of the right tibia and in the lumbar and dorsal vertebræ. The pain had become so severe as to cause serious disability. No improvement under antiluetic treatment.

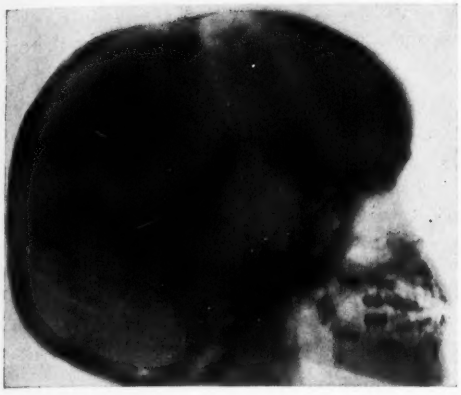


Fig. 55. See general caption which applies to case under Figures 51 and 52.

Fig. 56. Cyst- and tumor-forming metaplastic malacia. There are diffuse hyperostotic changes in the skull and an expanded tumor area in the frontal bone, near the bregma. (See Figures 57 to 65.)

Physical Examination.—Patient of medium size. Some papillary eruptions on back, and slight swelling in roof of mouth. No deformity of spine, but tenderness at the lower border of scapula. No general glandular enlargement. Reflexes very active. At middle crest of right tibia a slight swelling.

X-ray Examination (in the latter part of 1905) revealed definite tumor-like masses, the most marked one being in the upper portion of the right tibia. The right tibia was operated on and a tumor removed with bone curette. Pathologic examination showed areas very closely resembling giant-cell sarcoma.

The lesion of the bone was slow in repairing. Iodid of potassium was administered to saturation, during which time the wound healed rapidly. The patient was given a brace to relieve her back. At the time of discharge from the hospital there was no pain. The weight increased and general health was good.

Four months later, there was a return of the pain in the dorsal region and in the left and right thighs. The entire bony skeleton was then radiographed, cystic areas being found throughout the entire skeleton, while both femora were sites of pathologic fractures through the cystic areas. Lesions were found in the following bones: on the right side, at the base of the middle phalanx, third finger; in the second and third metacarpals; in the os magnum; upper end of ulna; at the junction of the lower and middle third of the femur, with fracture; outer malleolus and head of the fibula, and in the middle third of the tibia. On the left side, in the proximal phalanx, fourth finger; second and third metacarpals; in the trapezoid; in the middle third of the ulna; middle and lower third of the femur, with fracture; fourth and fifth dorsal vertebræ, and fourth and fifth ribs.

The foci were primarily in the cortex. They showed as well-defined, sometimes very small, sharply circumscribed, round or oval holes. Besides these lesions there were spindle-shaped enlargements of the shaft, circumscribed and multilocular.

Ten years later, May 15, 1915, a radiographic examination showed the following condition: In general, in the various bones, even where no cysts or tumors were present, there was general thinning of the cortex, with irregular endosteal thickenings. There

was a large oval mass in the posterior portion of the frontal bone, evidently located in the diploëtic layer. It was multilocular, with well formed trabeculæ and suggested a

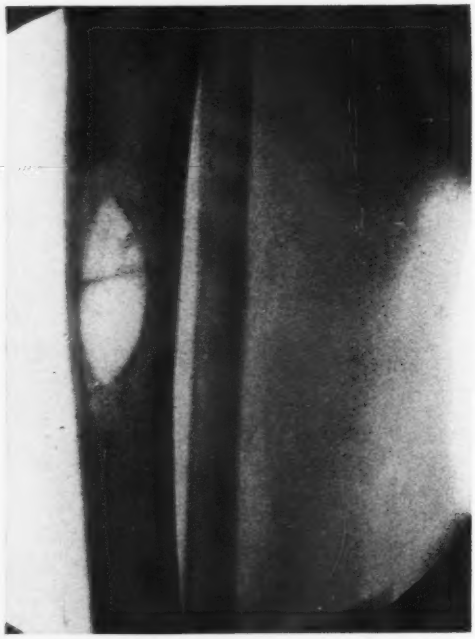


Fig. 57. Cyst formations in both tibiæ, some of which have ossified. There are metaplastic changes in many of the bones involved by tumors or cysts. (See Figures 56, 58 to 65.)

giant-cell tumor. There were lesions in the outer surface of the middle phalanx of the second finger (small cyst), in the proximal phalanx (ossified cyst), in the fourth metacarpal below its head, and in the fifth metacarpal. In the middle of the shaft of the radius there was a spindle-shaped enlargement due to a circumscribed trabeculated mass (giant-cell tumor). There were cysts and ossified cysts in the middle of the shaft of the ulna, in the middle third of the tibia, in the head of the tibia, and in the upper end of the fibula. The right femur showed an irregular fracture two inches below the lesser trochanter, through a multilocular cystic area, and there were cysts in the lower part of the shaft.

On the left side there were lesions in the second and fifth metacarpals and in the upper end of the ulna. There was an irregular oblique fracture of the femur at a point above the middle shaft, with mal-union; the cortex was thinned and the medulla widened. There was an ossified oval mass in the lower third of the tibia.

On the general background of diffuse osteitis fibrosa there existed numerous giant-cell tumors, some of which healed spontaneously and others after fracture had taken place through the involved area. The general condition remained good.

Similar changes may be found on the background of a diffuse osteitis fibrosa of the hyperostotic type. In other words, a hyperostotic metaplastic malacia, with tumor formations of the giant-cell variety.

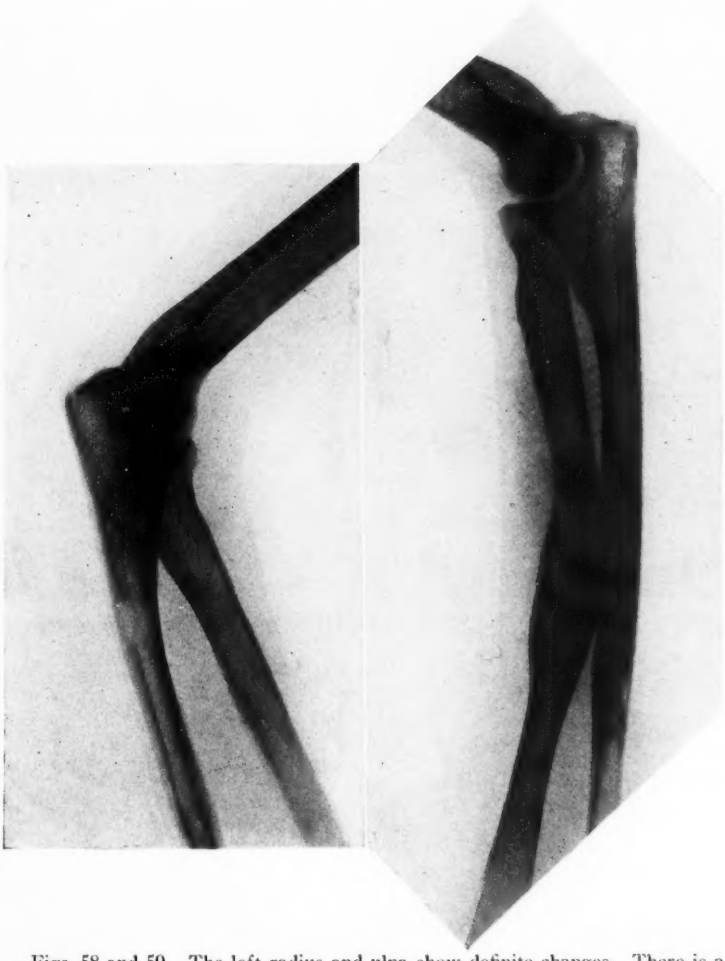
CONCLUSIONS

The pathologic relationships of the various types of malacia, as classified by von Recklinghausen, have been clearly identified on the basis of roentgen findings. The essential pathologic phases in all the types of generalized osteitis fibrosa give definite roentgen findings, and may be readily isolated.

The period of onset of generalized osteitis fibrosa has been brought back to the earliest period of life on the basis of the records in the literature of instances of early discovery, of hereditary tendencies, and of new cases reported and demonstrated. The following cases have been reported: (1) A case of cystic metaplastic malacia (von Recklinghausen), with a history of disease dating back to early childhood, which was observed over a period of fifteen years. (2) Three cases of hyperplastic malacia in three children in one family, the changes having existed from birth. (3) A case of combined metaplastic and hyperplastic malacia, in which the period of onset dates from child-

hood. (4) Two cases of cystic metaplastic malacia of the von Mikulicz type. (5) A case of cystic metaplastic malacia, with hyperostotic changes, which is clearly congenital. (6) Diffuse tumor-forming metaplastic malacia, with metaplastic newgrowths, in a girl of 22, the bones showing old, healed lesions which must have existed in childhood as judged by a comparative study over a period of ten years.

On the basis of this evidence, it is justifiable to infer that there are marked hereditary tendencies in generalized osteitis fibrosa and that in many cases the lesion already exists in early childhood or infancy. It is, therefore, logical to conclude that generalized hyperplastic, metaplastic and metaplastic hyperostotic malacia, with all their intermediary forms, are congenital hereditary diseases.



Figs. 58 and 59. The left radius and ulna show definite changes. There is a diffuse lamellation of the cortex and several small oval cystic areas in the cortex of the ulna. In the middle of the right radius is a sharply circumscribed, large, oval, multilocular, expanded area (giant-cell tumor). The shaft is markedly bowed at this point. In a corresponding area in the ulna, there is thickening of the cortex and minute cyst formations.

If this be true, then osteitis fibrosa may not be a metaplastic process (one tissue being transformed into another to which it is genetically related) incited by a causative factor generated *de novo* in the body. It

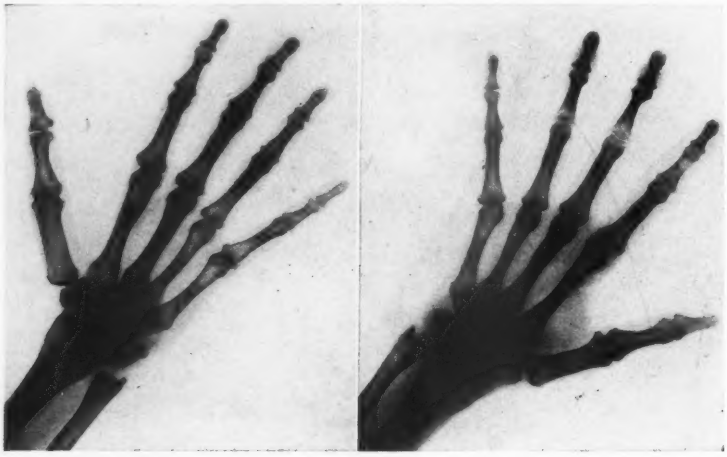


Fig. 60. Hands, showing fibro-osteoid cystic and localized cortical hyperostotic changes.

may arise from the osteogenetic cells of the periosteum and endosteum, due to developmental factors, to a congenital maldevelopment or weakness of the mesenchymatous tissues, much as osteogenesis imperfecta is a congenital disease due to deficient endosteal and periosteal bone formation arising from congenital weakness of the mesenchymatous tissues.[10]

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[10] Dawson and Struthers state that the cell content of the larger haversian spaces where the earliest stage of fibrous tissue substitution is taking place reveals a picture which presents many similarities to that seen in osteogenic development. Maximow has shown that early undifferentiated mesenchyme cells have a structure which can be recognized through the various stages of cell differentiation to the formation of osteoblasts and osteoclasts. This transformation has been observed in the genesis of osteitis fibrosa, where, however, the great mass of mesenchyme cells also were set apart to form fibrous tissue, which later completely replaced the resorbed bone.

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Fig. 61. Cystic changes in the femora and fractures, with deformity, through cystic areas.

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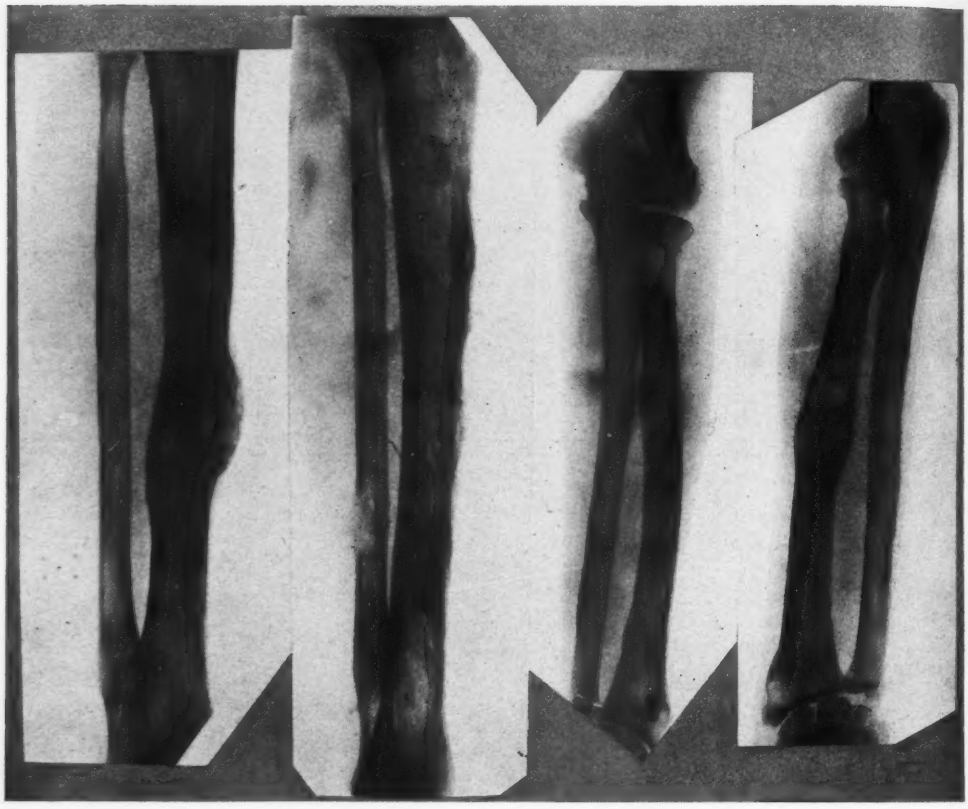

Fig. 62. Combined metaplastic and hyperostotic malacia, with tumor formation. Above the multilocular cyst formation, with expanded cortex, in the diaphyses of the tibia, is an area of diffuse bone thickening which is evidence of repair.

Fig. 63. Right tibia and fibula showing the diffuse involvement. A healed cyst can be seen in the upper end of the tibia. There are cortical cysts in the lower end of the fibula.

Figs. 64 and 65. Every pathological phase of generalized osteitis fibrosa can be found in the bones of the forearms.

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Fig. 66. There is general diffuse thickening of the vault of the skull, more marked, however, over the frontal area. The base is not involved.

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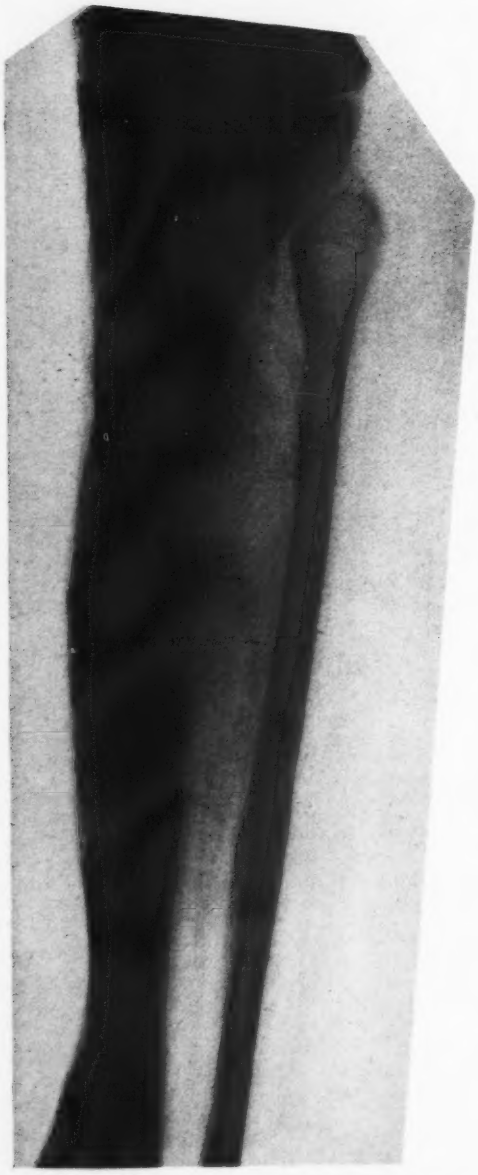

Fig. 67. Hyperostotic form of generalized osteitis fibrosa of the tibia. Note the arterio-sclerosis.

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Fig. 68. Myeloplastic malacia and rickets. There is to be noted the osteoporosis, the widened homogeneous medulla, and a thinned and striated cortex. The changes of rickets are also present to a marked degree. This radiograph might be said to illustrate the appearance of diffuse fibro-osteoid change before any reconstruction.

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DISCUSSION

DR. HIRSCH (closing): The purpose of the classification I have attempted to define is to simplify the comprehension of the large group of diseases which bear a definite relationship to each other but which until now have been considered as isolated entities. No one can fairly take exception to such an attempt. A simple classification makes for a better understanding, for more accurate diagnosis. The classification that I have attempted to elucidate may not be perfect, but at least it may serve as a working basis. We are in a position, as roentgenologists, to really contribute valuable data to the understanding of this group of diseases, and with this classification as a working basis I am sure that we shall arrive at a truer conception of the actual pathogenesis of the lesions of generalized osteitis fibrosa.

EDITORIAL

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THE ROENTGENOGRAPHIC FILM LIBRARY

The subject of forming a roentgenographic film library has been before the Radiological Society for several years. Experience in carrying out this project presented many difficulties. The methods used at first were costly in time, effort, and money. The suggestions that came from members of the Society increased the difficulties and presented problems that at the time were impossible of solution.

Through the annual scientific exhibit the subject has been kept alive; loan exhibits for the annual meeting have improved each year, and the exhibit has been instructive and interesting to a large proportion of the members. The keen interest and quest for information at these annual exhibits more and more made evident the necessity for a national library of films that shall be available to all members of the profession interested in roentgenology.

In developing a roentgenographic film library in The Mayo Clinic, methods have been devised which offer a solution of many problems.

In the majority of lesions of bone the original film is framed in a cardboard mount of a standard size, 8 by 10 inches. Many roentgenograms that are 10 by 12 inches or 14 by 17 inches in size can be cut to fit the standard mount without loss of proportion. All films of the thorax, and a small number of other films, have to be reproduced. A reduction is made on a 5 by 7 film, and from this a second reproduction is made on an 8 by 10 inch film. This double process brings the reproduction back to the values of the negative, the same as the original film.

An aperture of 7 by 8 inches in the cardboard mount will fit the greater number of films; a few require an opening 7 by 6½ inches. Alterations of the size of the aperture may be required in special types of studies. The outside measurement of 8 by 10 inches is maintained, as all view boxes are made to accommodate this size. Plain cardboard or chipboard answers for the rear mat; the front mat is of cardboard with a black facing. Both mats have a thickness of approximately 1 mm., to provide protection from scratching on both sides when films are piled or stacked together.

The upper and two side margins are ⅞ inch wide, and the lower margin 1¼ inch wide. A printed legend can be pasted on this lower border, or an opening of 6 by ¾ inch can be provided for a transparent legend. A transparent legend is made by typing the text on white, unglazed paper, photographing this, and cutting the film to the appropriate size.

The film, and the legend if a transparent legend is used, are fixed to the rear mat by a small metal fastener. The Bostitch fastener has been the most useful in our experience. The front mat is laid over this, and the mats are bound together by an aluminum strip which overlaps both margins by ⅜ of an inch.

Information has been received that all the material and apparatus to make up a film

library by this method are now available from one manufacturer.

Framing the original film has many advantages. The detail is preserved; the expense of reproduction is obviated; the initial step, that is, clipping the film to the rear mat and writing the legend, can be done at once and the rest of the process can be carried through by non-medical personnel. This allows, as a routine, segregation of interesting material for teaching, for study and other purposes. A reference note in the regular file indicates the situation of the film in the segregated file.

By the use of this method many of the difficulties of establishing the film library of the Society are solved. It is suggested that all members make up their exhibits for the 1929 annual meeting in this form. Arrangements are already under way for provision of new view boxes to accommodate this type of film. An easy and inexpensive way to amass material would be to take an extra set of films for mounting on every case of outstanding interest. At the annual meeting, a committee of judges may be appointed to pick outstanding films for the film library of the Radiological Society.

The ultimate aim should be the establishment of a national roentgenographic library, and an attempt to interest the American Medical Association in housing and administering this collection should be made by the officers of the Society. It is possible that universities, clinics and other similar organizations could be interested in donating sets of films to remain as a permanent memorial to the work of individual roentgenologists. Individual roentgenologists should be impressed with the idea that it would be a matter of pride to have achieved the honor of having had their film studies accepted for the national library. The usefulness of the Society to the profession in general and to its individual members could be greatly enhanced by the development of such a collection.

The individual members of the Radiological Society can do much to heighten the proficiency of roentgenologic diagnosis by hearty co-operation in the attainment of such an ideal.

CHARLES G. SUTHERLAND, M.B. (Tor.)

RADIOSENSITIVITY OF BONE[1]

Because of its hardness and strength, to which characteristics it owes its adaptation to the physiologic function it has to perform, and because of the length of time it can withstand decay and other destructive agencies, bone is commonly considered the most resistant tissue of the body. It is, however, only as properties of dead material that the hardness and strength and resistance of bone are apparent. As a living tissue, bone yields readily to constant pressure, even of softer tissues, by undergoing atrophy and resorption, and it may be quickly destroyed in inflammatory and neoplastic processes.

Resistance to radiation has also been ascribed to bone. Older tables of roentgen-ray dosages gave for bone a dose as much as eight times that for the skin of the face. With widespread use of the more deeply penetrating hard roentgen rays, bone has been found to be not immune to injury. Ewing[2] has described a form of chronic osteitis following irradiation, a proliferative process which is probably secondary to harm done rather than a primary stimulative reaction. More recently, Wynen[3] has discussed the occurrence of late necrosis in

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1Reprinted by permission from the *Journal of the American Medical Association*, April 27, 1929, XCII, 1447.

2Ewing, James: Radiation Osteitis. Acta Radiol., 1926, VI, 399.

3Wynen, W.: Die Radiosensibilität des Knochens in ihrer Bedeutung für die Röntgenbestrahlung der Gelenktuberkulose. München. med. Wchnschr., Feb. 8, 1929, LXXVI, 244.

bones subjected to therapeutic irradiation by roentgen rays. The process has been noted in the jaw and in bones connected with the exterior by fistulous openings; in the first case in bone easily invaded by bacteria after harm has been done, and in the second case in bone that is already infected but further injured by radiation. Experimentally it was found by Wynen that the rootlets of seedlings were more severely injured when surrounded by bone and subjected to radiation than when surrounded by an equally thick layer of muscle, an action ascribed to the emission of secondary rays by the calcium of the bone. Similar secondary radiation, resulting in the death of individual bone corpuscles and in injury to the capillaries, is held responsible for the harm to bone that may result in late necrosis under the further influence of bacterial invasion. Too intensive irradiation of bone apparently introduces an element of danger which must be borne in mind in the application of the therapeutic procedure. Wynen warns especially against too heavy dosage in the treatment of bone and joint tuberculosis.

Similar sensitivity of bone to radiation has been noted by Martland and his collaborators[4] as the result of the action of radio-active substances deposited in the bones of painters of luminous dials. In these unfortunates, not properly warned or protected against the hazards of a new occupation, widespread necrosis of the jaw occurred. Here, also, as is the case after roentgen irradiation, secondary bacterial invasion of bone injured by radiation is a factor. The injury to bone in dial painters is much more severe than has yet been described after roentgen irradiation, because the action of the deposited radium and mesothorium compounds is continuous over a period of years, the bones of a body exhumed after five years of interment having been found to be strongly radio-active by St. George, Gettler, and Muller.[5] Such continuous action leads not only to bone necrosis but also to diffuse radiation osteitis and to injury to the hematopoietic system, the latter manifested by an anemia of either regenerative or aplastic type, which is one of the principal features of the effects of radio-active substances. As still another possibility in the chain of evils caused by the deposition of radio-active materials in bone, Martland's[6] most recent contribution calls attention to the occurrence of osteogenic sarcoma of bone in two dial painters. The proof that the tumor was actually caused by the presence of radio-active substance in the bone is not and cannot be absolute. About 70 per cent of the deposited material is mesothorium, the half-life of which is six and seven-tenths years. If the victim escapes death from bone necrosis or anemia during the early period of more intense radio-activity, he still remains subjected to a less intense degree of activity over a prolonged period. Such long-continued but milder irritation may well be the cause of tumor formation, a possibility deserving consideration in the future history of those subjected to the intake of radio-active materials that may be deposited in the osseous system.

RADIUM RESEARCH

Uranium being always associated with radium, information about it is ever of interest to the radiologist. Hence we are pleased to observe that a new and extremely delicate chemical test for it has been discovered by Dr. Clifford Mitchell, of Chicago. The reagent used is sodium-alizarin-sulfonate and

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[4]Martland, H. S., Conlon, Philip, and Knef, J. P.: Some Unrecognized Dangers in the Use and Handling of Radio-active Substances. Jour. Am. Med. Assn., Dec. 5, 1925, LXXXV, 1769.

[5]St. George, A. V., Gettler, A. O., and Muller, R. H.: Radio-active Substances in a Body Five Years after Death. Arch. Path., March, 1929, VII, 397.

[6]Martland, H. S., and Humphries, R. E.: Osteogenic Sarcoma in Dial Painters Using Luminous Paint. Arch. Path., March, 1929, VII, 406.

the complete technic of the method is described in an article by Frederick G. Germuth, of Baltimore, research chemist in the Department of Public Works of that city, and Dr. Mitchell, published in the *American Journal of Pharmacy* for January, 1929.

Compounds of uranium in aqueous solution yield with a half of 1 per cent solution of sodium-alizarin-sulfonate a precipitate of distinctive deep violet color, insoluble in 1 per cent solution of acetic acid. By the aid of the colorimeter the violet color alone may be seen in remarkably dilute solutions. The reagent is useful also in the detection of a number of other metallic compounds.

FOURTEENTH ANNUAL SPRING MEETING OF THE ROENTGEN-RAY SOCIETY OF CENTRAL PENNSYLVANIA

The Fourteenth Annual Spring Meeting of the Roentgen-ray Society of Central Pennsylvania was held at the Hotel Dimeling, at Clearfield, Pa., Thursday, May 16, 1929. Sixty-five per cent of the membership were present. The total registration was 208, which included physicians from Clearfield and adjoining counties.

The name of the organization was changed to the Pennsylvania Radiological Society, the better to indicate the scope of its membership, which includes physicians from the entire state of Pennsylvania and several associate members from adjoining states.

The new officers are as follows:

President, Dr. J. M. Keichline, Huntingdon, Pa.

First Vice-president, Dr. William Levering, Stroudsburg, Pa.

Second Vice-president, Dr. A. L. Benson, Philipsburg, Pa.

Secretary-Treasurer, Dr. W. E. Reiley, Clearfield, Pa.

Editor, Dr. G. D. Bliss, Altoona, Pa.

The Fall Meeting will probably be held in Stroudsburg, Pa.

PROGRAM OF THE SECTION ON RADIOLOGY OF THE AMERICAN MEDICAL ASSOCIATION

FOR THE MEETING IN PORTLAND, OREGON, JULY 10, 11, AND 12, 1929

Officers of Section: Chairman, M. J. Hubeny, M.D., Chicago, Ill.; Vice-Chairman, Leon T. Le Wald, M.D., New York; Secretary, Fred M. Hodges, Richmond, Va.

Length of Papers and Discussions: The time allowed for the presentation of a paper before a section shall be limited to fifteen minutes. No one shall discuss any paper more than once, nor for longer than five minutes, except with the unanimous consent of those present.—*By-laws, Chapter XIV, Sec. 11.*

First Day—July 10

"Fundamentals of Roentgen-ray Treatment"
H. J. Ullmann, M.D., 1520 Chapala St., Santa Barbara, California.
Discussion to be opened by
G. M. MacKee, M.D., New York City.

"Fewer Variable Factors of Technic in Roentgen Therapy"
A. W. Erskine, M.D., Higley Bldg., Cedar Rapids, Iowa.
Discussion to be opened by
E. C. Ernst, M.D., St. Louis, and U. V. Portmann, M.D., Cleveland.

"End-results after Radiation of Fibromyomata"
Francis C. Wood, M.D., St. Luke's Hospital, New York City.

Discussion to be opened by
HENRY SCHMITZ, M.D., Chicago.

"Radiotherapy of Chronic Tonsillitis and Pharyngitis"
LEILA CHARLTON KNOX, M.D., New York City.
Discussion to be opened by
JOSEPH ASPRAY, M.D., 422 Riverside Drive, Spokane, Wash.

"Radiation in the Treatment of Carcinoma of the Breast"
G. E. PFAHLER, M.D., and LEO D. PARRY, M.D., University of Pennsylvania, Philadelphia.
Discussion to be opened by
R. C. BAUMGARTEN, M.D., Seattle, Wash., and ROY A. PAYNE, M.D., Portland, Ore.

"Radiation Service for the Modern Hospital"
ALBERT SOILAND, M.D., and O. N. MELAND, M.D., The Albert Soiland Clinic, 1407 S. Hope St., Los Angeles, Cal.
Discussion to be opened by
C. J. JOHANNESSON, M.D., Baker Bldg., Walla Walla, Wash., and H. B. THOMPSON, M.D., Seattle, Wash.

"Histological Changes in the Skin Following Irradiation"
E. A. POHLE, M.D., and C. H. BUNTING, M.D., University of Wisconsin, Madison, Wisconsin.
Discussion to be opened by
W. E. CHAMBERLAIN, M.D., Stanford Hospital, San Francisco.

Chairman's Address
M. J. HUBENY, M.D., Chicago, Ill.

Second Day—July 11

Joint Meeting with the Section on Gastroenterology and Proctology.

Clinicians—

"Malignant Lesions of the Lower End of the Esophagus and Upper Portion of the Stomach"
MAX EINHORN, M.D., New York.
Discussion to be opened by
J. M. BLACKFORD, M.D., Seattle, Wash.

"A Consideration of Chronic Ulcer of the Lower Esophagus and Upper Third of the Stomach"
F. A. SPEIK, M.D., Los Angeles.
Discussion to be opened by
FRANK SMITHIES, M.D., Chicago

Roentgenologists—

"Malignant Lesions of the Lower End of the Esophagus and of the Cardiac End of the Stomach"
P. M. HICKEY, M.D., University Hospital, University of Michigan, Ann Arbor, Mich.
Discussion to be opened by
B. H. ORNDOFF, M.D., Chicago

"Non-malignant Lesions of the Lower End of the Esophagus and of the Cardiac End of the Stomach"
A. B. MOORE, M.D., Mayo Clinic, Rochester, Minn.
Discussion to be opened by R. R. NEWELL, M.D., Stanford University Hospital, San Francisco.

Third Day—July 12

"Accidental Pneumothorax, with Report of Two Cases"
JOSEPH L. HARVEY, M.D., University of Colorado, Denver, Colo.
Discussion to be opened by
KENNETH ALLEN, M.D., Denver, Colo.

"Carcinoma of the Lung"
LLOYD BRYAN, M.D., Fitzhugh Bldg., San Francisco.

Discussion to be opened by
F. H. RODENBAUGH, M.D., and HAROLD BRUNN, M.D., San Francisco.

"Broncho-sinusitis Disease"
W. W. WASSON, M.D., 246 Metropolitan Bldg., Denver, Colo.
Discussion to be opened by
T. E. CARMODY, M.D., and T. D. CUNNINGHAM, M.D., Denver

"Esophageal Orifice Hernia"
MAX RITVO, M.D., 485 Commonwealth Ave., Boston, Mass.
Discussion to be opened by
A. L. GRAY, M.D., Richmond, Va., and E. P. RICHARDSON, M.D., Boston, Mass.

"Diverticula of the Stomach, with Report of Five Cases in which the Diagnosis has been Verified at Operation"
Ross GOLDEN, M.D., New York City, and J. C. BELL, M.D., Louisville, Ky.
Discussion to be opened by
E. S. ALLEN, M.D., Louisville, Ky.

"The Roentgenologic Diagnosis of Pedunculated Tumors and Gastric Mucosa Prolapsing through the Pylorus into the Duodenum"
E. P. PENDERGRASS, M.D., University Hospital, Philadelphia.
Discussion to be opened by
E. L. ELIASON, M.D., Philadelphia.

"Roentgenographic Characteristics of Bone Lesions"
C. G. SUTHERLAND, M.B. (Tor.), Mayo Clinic, Rochester, Minn.
Discussion to be opened by
W. B. BOWMAN, M.D., Los Angeles.

"Experimental Biophysics of Normal and Abnormal Bone Growth"
EBEN J. CAREY, M.D., Marquette School of Medicine, 638 Fourth St., Milwaukee, Wis.
Discussion to be opened by
LEMUEL D. SMITH, M.D., Milwaukee.

THE ADVISORY COMMITTEE ON X-RAY AND RADIUM PROTECTION

The Committee is composed of the following members:

L. S. TAYLOR, Bureau of Standards, *Chairman.*

W. D. COOLIDGE, PH.D., General Electric Co., Schenectady, N. Y.

H. K. PANCOAST, M.D., Philadelphia.

G. FAILLA, D.SC., New York City.

R. R. NEWELL, M.D., San Francisco.

J. L. WEATHERWAX, M.A., Philadelphia.

W. S. WERNER, of the Kelley-Koett Mfg. Co., Covington, Ky.

This Committee is representative of all radiological bodies in the United States and of all scientific and commercial interests. Its purpose is to consider problems in safety in this country, and its findings will be incorporated in the report of the International Committee at Paris in 1931.

A MEMBER OF THE SOCIETY RECEIVES FRENCH DECORATION

Dr. Amédée Granger, of New Orleans, has been awarded the Gold Palmes Universitaires by the French Government, an honor which confers the title of Officer of Public Instruction on the recipient. Dr. Granger is Director of the X-ray Department of Charity Hospital and Director of the Department of Radiology at Tulane University. He joined the X-ray staff at Charity Hospital in 1905, four years after graduation from Tulane University, and became Director when the department was reorganized in 1921. His service with the teaching staff of Tulane University dates back to

1908. He was appointed Professor of Radiology in 1916, and in 1925 head of the new Department of Radiology and a member of the executive faculty. In 1918 he instructed medical officers of the United States Army at Charity Hospital in the localization of foreign bodies.

The Palmes Universitaires (silver) was awarded Dr. Granger by France in 1921, followed in 1926 by an award from the Radiological Society of North America of its gold medal. In 1927, he received first prize for the best individual scientific exhibit at the Radiological Society's annual meeting. Alpha Omega Alpha, honorary medical fraternity, presented him with the key of honorary membership. Dr. Granger is a member of the College of Radiology.

THE PRECISE MEASUREMENT OF X-RAY DOSAGE

By LAURISTON S. TAYLOR, Associate Physicist, National Bureau of Standards, Washington, D. C.

In 1927, Behnken, of the Physikalische Technischen Reichsanstalt, made a comparison of the r-unit between several laboratories in Germany and those of Glasser and Duane. As a result of his measurements a difference of about 4 per cent was indicated between certain of the laboratories. As an outcome of his attempted comparison with the Bureau of Standards, studies were undertaken to determine the error due to the use of different methods for measurement of the ionization current.

A kenotron-rectified constant potential generator was used to supply the X-ray tube. The Duane parallel plate ionization chamber was used with several different electrical measuring systems, in order to study the differences existing among them. It was found that only a nul measuring method or the equivalent, such as a galvanometer, would yield accurate results. A simple deflection method or an idiostatic method distorts the field within the chamber in such a manner as to make the ionization current readings too high or too low, depending upon conditions. The use of a galvanometer (Duane) does not disturb the field sufficiently to cause any error.

A slight displacement of the ionization chamber measuring and guard electrodes may introduce an error. This is more serious in a deflection method than in a nul method. Ionization between the measuring electrode and case may affect the ionization readings in the idiostatic or deflection method, but not in the nul or galvanometric method.

The accuracy of the measurements can be carried out with a precision of 1.0 per cent or better, including the diaphragm error.

In the complete paper a full description of the *constant potential generator* is given.

There is also a full description and discussion of the nul electrostatic measuring method. (The complete paper appears in the *Bureau of Standards Journal of Research*, 1929, II, 771.)

BOOK REVIEW

TEETH AND JAWS ROENTGENOLOGICALLY CONSIDERED. By HERMAN A. OSGOOD, M.D., Roentgenologist to the Turnbull Hospital, Boston; Consultant Roentgenologist, Gale Memorial Hospital, Haverhill, Massachusetts. Second Edition, revised. Volume V, Annals of Roentgenology. Published by Paul B. Hoeber, Inc., New York, 1929. Pages 280. Price $10.00.

This edition contains several added features which make for a more complete and comprehensive consideration of this phase of roentgenology. Chapter IV contains a brief discussion of Dr. Howard Raper's bite wing film technic for the examination of crowns and pulp chambers of teeth, a technic which the dental profession is beginning

to recognize as one of the most important contributions to preventive dentistry in the past few years and as a final and satisfactory solution of the pulpless tooth problem.

A complete chapter has been devoted to the discussion of this problem. One gathers from reading it that the "roentgenogram cannot positively rule out the presence of infection in pulpless teeth," and if the roentgenologist knows very little, if anything, about the general condition of the patient he should not be called upon to advise the extraction or retention of pulpless teeth.

The author's discussion of the causes of peridontal disease is much more complete than in the first edition, and he has reclassified peridontal disease in accordance with the work of Box, Stillman, and McCall. The early identification of signs of traumatic occlusion or points of chronic irritation is the most important part of the roentgen examination.

A brief chapter on the roentgen ray in orthodontia points out that "the orthodontist in planning his procedure is enabled by the roentgenogram to study the condition of the deciduous teeth and their roots in relation to the permanent teeth below them, thereby obtaining information which cannot be obtained from models alone, such as the amount of absorption of the roots of a deciduous tooth, anomalies of decalcification or exfoliation, or other conditions necessitating the removal of a deciduous tooth to avoid deflexion of the permanent tooth below."

Subsequent roentgenologic examinations are recommended to check the progress of the case, watch for caries, and the evidence of too much trauma at the apex, thereby preventing injury to a tooth whose root is not completely formed.

The technical difficulties of making roentgenograms on the small intra-oral films for use in intra-oral stereoscopy are discussed in the chapter on "Methods of Localization and Intra-oral Stereoscopy," and a more satisfactory method than formerly used is introduced. A study of the films in the third dimension is of advantage to the exodontist, particularly pre-operative views of impacted third molars. The method of approach can be selected when one has determined the relation of the impacted tooth to the adjoining teeth or structures.

The entire book is well written and edited and covers its particular field of roentgenology very thoroughly. The roentgen-ray illustrations are enlarged and printed in the negative, which makes for easier reading and understanding by the average dental practitioner. One gathers from the legends accompanying these that the diagnosis was not made wholly from the roentgenologic findings.

ABSTRACTS OF CURRENT LITERATURE

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The Roentgenological Study of the Neck. Samuel Brown and Harold G. Reineke. Am. Jour. Roentgenol. and Rad. Ther., September, 1928, XX, 208.

The authors advocate a single lateral film of the cervical region in order to obtain useful information concerning the soft tissues in this region, particularly the pharynx, thyroid, and trachea. The normal relationship and relative dimensions of these structures are described, variations according to age and sex are considered, and a number of illustrations are included to show the abnormal displacements produced by retropharyngeal abscess, foreign bodies, and thyroid enlargements.

J. E. HABBE, M.D.

Cardiospasm, with Special Reference to Etiology. H. W. Soper and L. D. Cassidy. Am. Jour. Med. Sci., March, 1929, CLXXVII, 386.

This paper is based upon a study of 60 cases of cardiospasm, of which 29 revealed pathologic conditions in other organs. It appears that the nervous mechanism that controls the cardia may be disturbed by a large variety of pathologic conditions. The more closely the cases are studied, the more rarely will the diagnosis of idiopathic cardiospasm be made. Surgery is rarely indicated. The spasm yields readily to dilatation by either pneumatic or hydrostatic rubber balloon dilators.

ROBERT A. ARENS, M.D.

Our Experience on 25 Cases of Bone Sarcoma Treated by X-ray Deep Therapy. H. Holfelder. Strahlentherapie, 1928, XXXI, 33.

In the X-ray laboratory of the surgical clinic of the University of Frankfort-on-the-Main, 25 cases of bone sarcoma were treated during the years 1920-1921 and 1925-1926. Sixteen cases were observed longer than three years and 9 cases longer than two years following X-ray treatment. Of the first 16 cases, six were clinically cured, three of which were observed for more than five years. Of these 16 cases, three patients had a biopsy confirming the diagnosis; 10 died from the disease. Of the 9 cases which were only two years under observation, 7 are clinically cured so far, 6 of which had biopsy. In conclusion, it is stated that roentgen therapy of bone sarcoma should be given the preference over surgery.

E. A. POHLE, M.D., PH.D.

Radiation Therapy of Tumors of the Hypophysis. M. Nemenow and A. Jugenburg. Strahlentherapie, 1928, XXX, 239.

This is a report of 29 cases of tumors of the hypophysis: 18 patients had acromegaly (13 males, 5 females), 11 patients presented the adiposogenitalis dystrophy. The blood sugar of all patients was as a rule below normal, while the calcium in the blood was higher. It reduced, however, with the improvement in the condition. In all except one

case, there was marked improvement. The headaches disappeared and the disturbance in the vision was relieved. There was also considerable psychic improvement. All patients except two were capable of earning their own living. In women, the menstruation returned after it had disappeared in the course of the disease. Technic: 4 or 5 fields, 6 × 8 cm., 200 to 220 K.V., 5 Zn. + 3.0 Al., 80 per cent E.D. In some cases radium was given in one to two millimeter platinum tubes of 20 milligrams each and left in the nose for ten hours. The second series of treatments was given after a three months' interval, after the hair had regrown.

E. A. POHLE, M.D., PH.D.

Roentgen Studies of the Bones in Certain Diseases of the Blood and Hematopoietic System. Rolla G. Karshner. Am. Jour. Roentgenol. and Rad. Ther., November, 1928, XX, 433.

Bone changes are rarely demonstrable by the X-ray in the anemias and allied diseases, but when they do occur are often of assistance in making the diagnosis.

In von Jaksch's anemia the cranium shows an increased thickness of the medullary space at the expense of a thinning of the tables. In the late stages one may see striations perpendicular to the tables. The bones of the body may appear very porous, with a thinned cortex and transparent medulla.

In Gaucher's disease no skull changes occur but one frequently sees destructive joint lesions, particularly in the hips, wedge-shaped narrowing of many bodies of the vertebræ without changes in the interspaces, periosteal elevation of the long bones, and spontaneous fractures.

In the leukemias one may see on the X-ray film periosteal elevation due to lymphatic infiltration, osteosclerosis, small or extensive foci of bone destruction, a moth-eaten appearance of the medulla suggesting decalcification, or a generalized decrease of bone density.

The X-ray findings in chloroma resemble closely those observed in cases of leukemia, except that there may also be a widening of the cranial sutures.

Hemophilia shows typical areas of calcification within the joint, associated with punched-out areas of destruction at the articular edges.

Purpura hemorrhagica shows no demonstrable X-ray changes.

J. E. HABBE, M.D.

On the Relation of Gall-bladder Emptying to the Ingestion of Fats: II. D. N. Silverman, Willy Denis, and H. L. Weinberger. Am. Jour. Med. Sci., March, 1929, CLXXVII, 384.

The writers made numerous experiments on the reaction of the gall bladder to various fats and emulsions. They found that the gall bladder empties only slightly after unemulsified fat, as olive oil; that egg yolk produced a more rapid and complete emptying than did cream. They also came to the conclusion that the emptying of the gall bladder is apparently not dependent upon fat absorption as far as can be determined by blood analysis. Also that there is suggested some relationship of gall-bladder emptying and intestinal digestion of fat.

ROBERT A. ARENS, M.D.